Alcohol, Tobacco and Cannabis as Factors in the Perpetration of Violence Across Adolescence and Early Adulthood

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SUMMARY

I investigated the longitudinal relationship between alcohol use, cannabis use and cigarette smoking and serious violence using data from a prospective longitudinal, nationally representative cohort of 17,519 individuals interviewed on 4 occasions. Participants were between 12 and 17 years of age at wave I and were between 23 and 32 year of age at wave IV.

There was a linear relationship between the number of drinks consumed but not frequency of drinking alcohol and violence. The number of individuals needed to abstain from drinking alcohol to prevent one from becoming violent was estimated. Smokers were also twice as likely to report subsequent violence within the next year, however there was no relationship between cannabis use and incident violence within the next year.

Analysis of the entire cohort (whether or not they reported violence at baseline) incorporated individual change in substance use over time to investigate the longitudinal relationship between substance use and violence. Moderate drinkers were approximately 1.4 times more likely to be violent than non-drinkers. Cigarette smoking and cannabis use was also associated with similar increases, but heavier drinkers were more than twice as likely to report serious violence. When the trajectories of violence were investigated, predictive marginal effects showed that drinking 1-4 drinks on each occasion was associated with an increased risk of violence during adolescence, but as the individuals got older the risk of violence tended to converge with that of non-drinkers by age 20. Heavier drinking however, was associated with a greater risk of violence well into adulthood, although the trajectories tended to converge by age 30.

The relationship between personality traits, alcohol and violence was then investigated using structural equation modelling. Alcohol was found to mediate the association between violence and specific personality characteristics, especially anger/hostility and extroversion.

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OVERVIEW OF THESIS

The longitudinal relationship between the most common substances of abuse - alcohol, cannabis and cigarettes - and serious violence was investigated using data from a prospective longitudinal, nationally representative cohort of 17,519 male and female adolescents first interviewed in 1994-95. Data collection interviews took place in four waves, the most recent in 2008. Participants were between 12 and 17 years of age at wave I, between 13 and 18 years at wave II, between 18 and 26 years at wave III, and between 23 and 32 years of age at wave IV.

Section 1

The relationship between alcohol use at Wave I and incident violence at Wave II was analysed. A comprehensive set of confounding variables was investigated. It was found that the most important confounders were age, delinquency, cigarette smoking and peer substance use. Ethnicity, IQ, depression, impulsivity, temper and neighbourhood disadvantage were also important, but to a lesser extent. After adjusting for confounders, the odds of violence within one year after drinking alcohol was about one and a half times higher than for those who were non-drinkers. There was also evidence of a relationship between the number of drinks usually consumed on each occasion and later violence; those who consumed 5-10 drinks were twice as likely to be violent at wave II, and those who drank 11 or more were 4 times more likely to be violent. There was evidence of a linear relationship between number of drinks consumed on each occasion, frequency of binge drinking and frequency of intoxication and violence, but not frequency of drinking *per se.*

The relationships between cigarette smoking and violence and cannabis use and violence were then investigated. After adjusting for confounders, I found that smokers were approximately twice as likely to report subsequent violence as non-smokers, however there was no linear relationship with number of cigarettes smoked. There was no relationship between cannabis use and subsequent violence after adjusting for confounders.

The number needed to prevent violence was calculated. Assuming that there was no residual confounding, and that the association was causal, it would be necessary for 54 people to abstain from drinking alcohol to prevent one of them from

becoming violent within the following 12 months (95% CI 23-671). In order to prevent one person from being violent, approximately 37 (range 17-189) would be required to change from usually drinking 5 or more drinks, to usually consuming less than 5 drinks each occasion when they drank, and 47 people (range 25-146) would be required to refrain from binge drinking at all.

Section 2

In this section I extended the previous analyses to investigate the relationship between alcohol, cannabis use or cigarette smoking exposure at baseline, and subsequent violence at waves II-IV among those not already violent at baseline, using a population average method, and whether there was a significant association between the length of time elapsed since wave I and violence. As before, those who drank 5 or more drinks at baseline were more than twice as likely to be violent than those who drank 4 or fewer drinks. There was also evidence of a linear relationship between frequency of binge drinking and of getting very drunk and subsequent violence, but not frequency of drinking alcohol. There was no evidence of an association with time or an interaction effect between exposure and time. This indicates that the risk associated with alcohol consumption at wave I is constant with respect to violence at either wave II, II or IV (independently of whether they continued to drink alcohol in the same quantity).

There was weak evidence that cigarette smoking was associated with subsequent violence (OR 1.51 for those smoking 1-60 cigarettes per month), but no linear trend. There was no evidence that cannabis use was associated with later violence.

Section 3

In contrast to the methods used previously, which considered average effects within the cohort with respect to incident violence, this section now considers the behaviour of each individual within the sample, and investigates differences between individuals over time. This approach accommodates individual change in exposure over time as well as individual differences in the outcome over time.

A comparison between those who were violent at baseline (and who were excluded from the previous analyses) and those who were not was first carried out. At baseline, more of those who were violent were male than those who were not violent; those who were violent at baseline were also more delinquent, had more

friends who used substances, were more depressed, had a lower mean IQ, were more impulsive, and came from neighbourhoods with a higher violent crime rate.

In random-effects models of the entire cohort, those who drank 1-4 drinks each occasion were 1.4 times (95% CI 1.2, 1.6) more likely to be violent than non-drinkers and those who drank 5 or more drinks just over twice as likely (OR 2.1, 95% CI 1.8,2.4) to be violent. . Regular smokers (average 2 cigarettes per day or more) were 1.5 times (95% CI 1-3-1.8) more likely to be violent. Cannabis use was also associated with violence (OR 1.4, 95% CI 1.20-1.70 among those using it up 1-10 times a month; OR 1.7, 95%CI 1.4-2.10 among those using it 11 or more times a month). Having been reported by a parent to have difficulty controlling temper at baseline was also shown to be significantly associated with violence (OR 1.6, 95% CI 1.4-1.7).

The trajectory for violence was found to be one that decreased with age; this held true whether alcohol, cannabis use or cigarette smoking was investigated. Predictive marginal effects showed that drinking 1-4 drinks on each occasion was associated with an increased risk of violence during adolescence, but the trajectory then tended to converge with that of non-drinkers. Drinking 5 or more drinks, however, was associated with a greater risk of violence well into adulthood, although the trajectories tended to converge by age 30. Regular smoking was also associated with a small increase in violence during adolescence, but the effect diminished with age, while the effect of cannabis use in relation with violence remained well into early adulthood.

Section 4

My finding of a reported difficulty managing one's temper being independently associated with violence prompted me to investigate personality traits. The relationship between personality traits, measured using the 5-factor model, alcohol and violence was then investigated using structural equation modelling. A model in which alcohol mediated relationships between personality and violence was found to fit the data better than one in which personality factors mediated relationships between alcohol and violence. Agreeableness was inversely associated with violence in both men and women (that is, being disagreeable was associated with violence). Alcohol mediated approximately 11% of the effect in males, but there was no evidence of an effect in women. Similarly, anger-hostility was strongly associated with violence in both sexes, but alcohol mediated the effect

only in males (approximately 20% of the total effect). Neuroticism was not associated with violence, and was associated with alcohol use only among men, but extraversion was associated with violence, with alcohol accounting for 15% of the effect in men and 29% in women.

Conclusions and Implications

Longitudinal associations between early alcohol consumption and later violence were found, even after controlling for a comprehensive set of confounders. Quantity of alcohol consumed each time rather than frequency of consumption is associated with violence, so measures to control binge drinking would be more effective in reducing violence than measures to reduce prevalence of consumption. The effect of alcohol consumption on violence was greatest the younger the individual, and gradually reduced with age and length of time since first consumption. Efforts to reduce alcohol related violence would be most effective in reducing heavy alcohol consumption among young teenagers.

Cannabis use and smoking appear to have a longitudinal association with violence and further research is required to ascertain the possible mechanism. As these relationships were not dose related, it seems less likely than for alcohol that there is pharmacological component/physical component to the explanation. The nature of the relationship between personality factors, alcohol misuse and violence differs between men and women to an extent that may be relevant for interventions. Reducing alcohol consumption in men but not women with disagreeable and angry/hostile traits would have a small but significant effect in reducing their risk of violence. For both sexes among those who have high extraversion ratings it would appear important to intervene with respect to alcohol consumption as well as personality style in order to reduce violence risk.

SECTION I INTRODUCTION

CHAPTER 1 EPIDEMIOLOGY OF VIOLENCE

DEFINITIONS OF VIOLENCE

The World Health Organisation defines violence as "*The intentional use of physical force or power, threatened or actual, against oneself, another person, or against a group or community, that either results in or has a high likelihood of resulting in injury, death, psychological harm, maldevelopment or deprivation*" (WHO, 1996). Given the diverse nature of violence encompassed by this definition, typologies have been developed to differentiate types of violence. Perhaps the most pragmatic is that which divides violence according to the characteristics of those committing the act: self-directed violence (including suicide and self harm); Interpersonal violence (violence inflicted by another individual or a small group of individuals); and collective violence (violence committed by larger groups such as states, and political groups or terrorist organisations)(Krug et al., 2002). Interpersonal violence has been further divided into family and intimate partner violence (violence mostly between family members and intimate partners, usually taking place in the home), and community violence (violence between individuals who are unrelated and may not know each other, generally taking place outside the home. The types of violent acts (with the exception of self-directed violence) can be further categorised into one of four categories based on the nature of violence; these categories are physical, sexual, psychological and deprivation or neglect.

THE BURDEN OF VIOLENCE IN THE GENERAL POPULATION

In 2000, an estimated 1.6 million people worldwide died as a result of violence, of which an estimated 520,000 were homicides (Krug *et al.*, 2002). The overall age-adjusted homicide rate was 8.8 per 100,000 population. The highest rates of homicide were found among males in the 15-29 year age-group (19.4 per 100,000). A recent meta-analysis showed that, globally, violence accounts for around 9% of young male deaths (Patton *et al.*, 2009).

Estimates of non-fatal violence are more difficult to obtain and rely on reports to police, self-report in surveys, and, where they exist, surveillance systems in medical centres. The Crime Survey for England and Wales, an annual survey of around 50,000 households, reported that in 2011/2012 there were approximately 2 million violence incidents committed against adults in England and Wales (Office for National Statistics, 2012a), resulting in 300,000 emergency department attendances and 35,000 hospital admissions (Sivarajasingam *et al.*, 2011). The highest rates of violence are found among youths; around half of all violence reported by adults was committed by 16-24- year olds (Flatley *et al.*, 2010).

Rates of violence are not globally distributed evenly. For example, In the USA 44% of adolescents reported physically fighting in the past year (Grunbaum *et al.*, 1999), compared with 24% in Sweden (Grunbaum *et al.*, 1999) and 76% in Jerusalem (Gofin *et al.*, 2000).

The overall cost of violence to society in the UK in 2012 was estimated to be £124 billion. This is equivalent to 7.7% of overall gross domestic product (GDP)(Institute for Economics and Peace, 2013).

VIOLENCE AS A PUBLIC HEALTH ISSUE

Since the 1970s, violence has increasingly been recognised as a public health issue, not just a social problem or a matter of law and order. A number of trends contributed to this development (Dahlberg and Mercy, 2009): first, the increasingly effective treatments of diseases which caused the greatest mortality in western countries (primarily communicable diseases) through public health measures resulted in violent deaths taking a larger overall proportion of mortality figures, thus increasing the prominence of violent deaths in national mortality statistics. Secondly, an increase in homicide rates, particularly in the USA and other developed countries, among young people and minority groups during the 1980s and 1990s to "epidemic" proportions (Centers for Disease Control and Prevention, 1994) raised political concerns and led to calls for new approaches to dealing with violence. Thirdly, it was becoming more accepted that behavioural and lifestyle factors are important in the aetiology of disease (such as diet or exercise in the prevention of cardiovascular disease), which can be successfully targeted by public health strategies. This led, by analogy, to the hope that violence could also be

tackled by applying public health interventions, once the risk factors for violence were understood.

During this period there were several important publications that emphasised the public health significance of violence (Dahlberg and Mercy, 2009). In 1979 a report from the Surgeon General in the USA stated that violence is preventable and listed it as one of the 15 priorities for intervention to improve the health of the nation. This was followed by the establishment of the Violence Epidemiology Branch of the Centers for Disease Control and Prevention in the USA. Subsequently in 1990, "violent and abusive behaviour" was included as one of twenty-two public health priority areas in a national disease and health promotion strategy (Office of Disease Prevention and Health Promotion, 1990).

A significant step in the international recognition of violence as a public health concern was made when the World Health Organisation adopted Resolution WHA49.25 in 1996. This resolution declared violence a leading worldwide public health problem, and urged member states to assess the problem of violence in their own territories, to initiate public health activates to define and assess the public health consequences of violence, to assess the effectiveness of violence prevention programmes, and to promote research on violence as a priority within a public health research framework.

Eight years later, in 2002, the World Health Organisation published The World Report on Violence and Health (Krug *et al.*, 2002), a report on the progress made following the adoption of Resolution WHA49.25. The stated goals of the report were to raise awareness of the problem of violence globally, and to make the case that violence is preventable and that public health has a crucial role to play in addressing its causes and consequences. It stated that the public health approach to tackling violence is underpinned by four key steps: (1) systematically collecting basic data on the magnitude, scope and characteristics and consequences of violence, (2) conducting research on the causes and correlates of violence, and identifying the factors that increase or decrease it and the factors that may be modifiable through intervention, (3) exploring ways to prevent violence using the information acquired by designing and implementing and monitoring interventions, and (4) implementing interventions that appear to be effective, widely disseminating information and determining he cost-effectiveness of programmes.

There followed calls for multidisciplinary work to address violence in medical literature, e.g. (Shepherd, 2002).

In the United Kingdom, the Department of Health published a comprehensive report on violence and public health in 2012 entitled "*Protecting people, promoting health. A public health approach to violence prevention for England*" (Bellis *et al.*, 2012), which explicitly set out the argument that violence, like disease, is preventable by applying a public health method. The report states (page 6): "*Many of the risk factors that make individuals, families or communities vulnerable to violence are changeable, including exposure to adverse experiences in childhood and subsequently the environments in which individuals live, learn and work throughout youth, adulthood and older age. Understanding these factors means we can develop and adopt new public health based approaches to violence. Such approaches focus on the primary prevention of violence through reducing risk factors and promoting protective factors over the life course*".

Although there is a multitude of factors that contributes to violence, the public health approach is to identify the causal factors systematically, so that strategies can be put in place to prevent violence. The contribution of alcohol to violence has received a great deal of attention as evidence for the association between alcohol and violence is present in many sources. Criminal statistics from several different countries show a high proportion of violent crimes are carried out by people who are under the influence of alcohol at the time of the offence. In England and Wales, for example, approximately 50% of victims of violent crime reported that the perpetrator was under the influence of alcohol at the time of the assault (CSEW, 2013). A history of alcohol problems is also very prominent among the perpetrators of crime. A systematic review of alcohol abuse histories of prisoners included 4,141 prisoners and found the prevalence of alcohol abuse or dependence ranged from 17.7% to 30% among men, and between 10% and 23.9% among women (Fazel *et al.*, 2006). Subsequent studies have found the rate to be even higher (e.g. (Kissell *et al.*, 2014) so a greater understanding of this relationship may contribute towards designing and targeting interventions that can be used to reduce violence. It is arguable here that, although it is likely to be important to treat convicted offenders for established alcohol misuse, a preventive approach would be even more worthwhile. Thus, investigation of alcohol as an early risk factor for violence may create scope for public health measures, which could lead to primary prevention of the violence.

In this study I have focused on the role of alcohol use by teenagers and young adults and subsequent serious interpersonal violence, which involves physical injury inflicted on another. I have made no explicit distinction between family and community violence and have included both, and have used the severity of violence as the main outcome of interest rather than the location of the violence or relationship between victim and perpetrator.

CHAPTER 2 ALCOHOL

EPIDEMIOLOGY OF ALCOHOL MISUSE

The functional ingredient of alcoholic beverages is ethyl alcohol (also called ethanol) and has a molecular formula C_2H_5OH. It is a clear, colourless liquid produced by fermentation, the process of transforming carbohydrates to ethyl alcohol by growing yeast cells.

Alcohol has been consumed by humans since antiquity; ancient myths, stone-age archaeological findings and numerous biblical references attest to the fact that alcohol has been part of tradition, ritual and custom for much of human history, at least as early as 10,000 BC (Patrick, 1952). Until approximately the beginning of 16th Century, fermented alcoholic beverages were consumed in all known village societies except Australia, Oceania, and North America (WHO, 2014). Alcohol production was typically small scale and seasonal depending on agricultural and seasonal availability of raw ingredients. Alcoholic beverages have been used for supposed medicinal purposes, in religious custom, to quench thirst and for social purposes. The process of alcohol production became industrialised, and the advent of distillation brought about products that could be more effectively stored and easily transported and traded as a commodity. The increased supply and availability brought about an increase in consumption often associated with severely detrimental effects on public health and indigenous economies. In the 19th Century in industrialised countries popular social movements to limit alcohol consumption gained momentum, followed by political will to control and in some cases prohibit alcohol consumption (WHO, 2014).

Today, alcohol consumption varies widely between countries, ranging from 1.9 litres of pure alcohol per person over the age of 15 per year in Islamic middle east countries and the Indian subcontinent to around 11 litres per person per year in Europe, North America, Australasia and Japan (Room *et al.*, 2005, Room *et al.*, 2002). Individuals in the former USSR, including the Russian Federation, Ukraine, Latvia, Estonia, and also Czech Republic and Luxemburg have an even higher alcohol consumption, estimated as over 15 litres per person per year (Ginter and Simko, 2009). Worldwide, it is estimated that 38% of people age 15 or over had consumed alcohol in the previous 12 months (WHO, 2014), however 48% of the

adult population has never consumed alcohol. In the UK, data from 2008 show that 87% of the population of England over 16 consumed alcohol at least once.(Fuller *et al.*, 2009).

The National Institute of Alcohol Abuse and Alcoholism (NIAAA) in the United States of America reported trends of alcohol use among young people age 12-20 between 1991 and 2011. The most recent data show that approximately 4% of 12-14 year olds, and a fifth of 15-17 reported drinking alcohol in the preceding 30 days with a similar prevalence in both males and females. The median age of first ever drinking was 14.3 years. The survey found that 15% of 12-20 year-olds reported binge drinking in the preceding 30 days (they did not break down the findings further by age).

There are geographical differences in the types of alcoholic beverages that people consume. Approximately one half of global alcohol is consumed in distilled spirits (WHO, 2014). Approximately 35% is consumed in beer, and 8% of recorded alcohol is consumed in wine.

The volume of pure alcohol in an alcoholic drink is measured in units of alcohol. One unit of alcohol contains 10ml or 7.9g of alcohol in the UK. The health risks associated with alcohol consumption increase with the amount consumed. The NHS in England and Wales have categorised the consumption of 21 units of alcohol per week (and no more than 3-4 units in a day) for adult men, and 14 units a week (and no more than 2-3 units per day) for adult women as "low-risk".

BURDEN OF ALCOHOL MISUSE

The World Health Organisation estimates that 5.9% of all deaths per year worldwide are attributed to alcohol, and 5.1% of the global burden of disease measured in disability-adjusted life years (DALYs)(WHO, 2014). Mortality and morbidity attributed to alcohol is from a range of medical disorders including the direct effects of alcoholism, liver disease, cancers of the gastro-intestinal system, heart disease, and pancreatic disease. Furthermore, 22% of deaths from interpersonal violence are attributed to alcohol.

CLASSIFICATION OF ALCOHOL MISUSE AND ALCOHOL USE DISORDERS

The international classification of diseases (ICD-10) has several diagnostic categories for mental and behavioural disorders due to psychoactive substance use – which can apply to alcohol, cannabinoids or tobacco use specifically as well as other classes of psychoactive substances. For alcohol, they are:

Acute alcohol intoxication – in 'uncomplicated' form, or complicated in various ways, for example by coma or delirium

Harmful use. This is defined as a pattern of substance use that is causing damage to health, either physical or mental. The diagnosis requires that actual damage is occurring.

Dependence syndrome. This is defined on the basis of three or more of the following criteria present together within a year:

A strong desire or sense of compulsion to take the substance;

Difficulties in controlling substance-taking behaviour in terms of its onset, termination, or levels of use;

A physiological withdrawal state when substance use has ceased or have been reduced, as evidenced by: the characteristic withdrawal syndrome for the substance; or use of the same (or closely related) substance with the intention of relieving or avoiding withdrawal symptoms;

Evidence of tolerance, such that increased doses of the psychoactive substance are required in order to achieve effects originally produced by lower doses (clear examples of this are found in alcohol- and opiate-dependent individuals who may take daily doses sufficient to incapacitate or kill non-tolerant users);

Progressive neglect of alternative pleasures or interests because of psychoactive substance use, increased amount of time necessary to obtain or take the substance or to recover from its effects;

Persisting with substance use despite clear evidence of overtly harmful consequences, such as harm to the liver through excessive drinking, depressive mood states consequent to periods of heavy substance use, or drug-related impairment of cognitive functioning; efforts should be made to determine that the

user was actually, or could be expected to be, aware of the nature and extent of the harm.

Withdrawal state is defined as a group of symptoms experienced after cessation of substance use after it being taken repeatedly over a prolonged period. It is one of the indicators of the dependence syndrome. In the case of alcohol withdrawal states, the symptoms usually appear 6-48 hours after cessation of alcohol consumption, and may include muscle ache, twitches, perceptual distortions, and may proceed to grand mal seizures and delirium tremens (see below).

Withdrawal state with delirium. This is a withdrawal state complicated by delirium. In the case of alcohol withdrawal, the condition is known as delirium tremens. It is a state characterised by clouding of consciousness, confusion, vivid hallucinations and marked tremor. It has been reported to be fatal in around 15% of cases, however improved treatment using benzodiazepines to detoxify has resulted in a much lower fatality rate(Griffin *et al.*, 1993).

Psychotic disorder. This is a psychotic disorder that occurs immediately after taking the drug (usually less than 48 hours). The disorder typically resolves at least partially within 1 month and fully within 6 months.

Amnesic syndrome. This is a syndrome associated with chronic impairment of learning new material including disturbance of time sense. It the case of alcohol abuse, the diagnostic category includes "Korsakov's syndrome", which is a neurological condition caused by thiamine deficiency causing damage to the medial thalamus and mammillary bodies in the hypothalamus, and generalised cerebral atrophy. The symptoms include severe memory loss (both anterograde and retrograde), apathy, and poverty of thought, confabulation and lack of insight.

In addition to the ICD-10 classification, several term are widely used that are not within the ICD-10 classification. They include:

Binge drinking. This is a term in popular use and in research studies to describe a pattern of episodic heavy drinking. However, there is no consistently agreed definition as to what constitutes binge drinking or how it can be accurately measured (Murgraff and Parrott, 1999). Two approaches have been used; the first is based on the number of units of alcohol consumed during a stated period. In the General Household Survey, ONS Omnibus survey and Health Survey for adults in England, and the Youth Lifestyles Survey (YLS), all UK government funded surveys,

binge drinking was defined as consuming 8 or more drinks for a man and 6 or more drinks for a woman (Richardson and Budd, 1993, Richardson and Budd, 2003). Many US-based surveys, however, have defined binge drinking as consuming 5 or more drinks in a row (Blitstein *et al.*, 2005). Add Health, the US database which I have use in my study, uses the latter definition.

Direct comparison between international surveys is hampered, not only because of different definitions of binge drinking, but also because of different definitions of what constitutes a standard drink. The UK has adopted a system of "units" of alcohol; each unit contains 10ml or 7.9g of ethanol. A pint of average strength beer contains 2 such units; a standard single measure of spirits contains 1 unit. Questionnaires in the UK therefore have to ensure that respondents are clear whether they are counting and reporting drinks or units. A standard drink in Austria contains 7.6 ml (6g) of ethanol and in Japan a standard drink contains 25ml (19.75g) of ethanol. In the USA, a standard drink contains 17.7ml or 14g of ethanol. Therefore a "binge" of alcohol would correspond to a smaller amount of alcohol in the USA compared with the UK.

The other method of defining binge drinking rests on the acute effect of intoxication. Here too, there is variation. For example, an extreme definition is continuous dependent drinking over a day or more until the drinker is unconscious (Newburn and Shiner, 2001), compared with more frequently used definitions such as getting intoxicated with alcohol at least once per week (Richardson and Budd, 2003). The definition of "intoxication" is also of course subjective.

Other categories that are not found within the ICD-10 classification are:

Increased risk drinking or **hazardous drinking** which is defined as regularly drinking 22-50 units of alcohol per week for men, and 15-35 units per week for women, and **Higher-risk drinking,** which is defined as drinking over 50 units of alcohol per week for men and over 35 units week for women.

CHAPTER 3 THE ASSOCIATION BETWEEN ALCOHOL AND VIOLENCE

In this chapter I outline the findings mainly from prospective longitudinal studies that have investigated the relationship between alcohol and violence. I highlight that there is mixed evidence from the studies, and discuss some of the possible reasons for the inconsistent findings. These include differences in definitions of violence, differences in statistical methods, and variation in the thoroughness with which confounding has been addressed. I then present a summary of the main risk factors that are common to both alcohol use and violence, which may prove to be confounders.

OVERVIEW OF ALCOHOL AND VIOLENCE

Evidence for the association between alcohol and violence has been drawn from many sources and from several different countries. Evidence shows that a high proportion of violent crimes are carried out by people who are under the influence of alcohol at the time of the offence (CSEW, 2013), that a high proportion prisoners have a have a history of alcohol problem (Kissell *et al.*, 2014), and general population surveys show a high proportion of people report a personal association between drinking alcohol and violence. In a sample of 602 Americans aged between 17 and 21, for example, who reported drinking in the last year, 40% reported that they had engaged in fighting after drinking (Wells *et al.*, 2006). Those who were younger were significantly more likely to report fighting, and there was a stronger relationship among males than females. General population based data also show a relationship between alcohol consumption and violent crime. A study from Norway investigated the relationship between rates of violence in the population and *per capita* alcohol consumption over more than one hundred and twenty years; the main findings was a positive relationship between alcohol consumption and violent crime, and an estimation that an increase in alcohol consumption by 1 litre per year per capita, would result in an 8% rise in violence in the population. The authors concluded that the data supports a causal model of alcohol on violence (Bye, 2007), however as an ecological study there may be problems with this interpretation due to confounding and ecological fallacy

(erroneous inferences about the nature of individuals made from correlations made for the group to which the individuals belong).

Although there is a wealth of evidence that shows that alcohol and violence are associated, there are several possible explanations for the observed association, other than a causative one. It is possible that both the propensity to acting violently and the propensity to alcohol misuse are different expressions of the same underlying syndrome of problem behaviours. Another explanation may be that the association is spurious due to confounding, that is that violence is due to other underlying causes which are also associated with alcohol. Alternatively, the explanation may be that violence causes alcohol misuse (reverse causality).

ARE ALCOHOL USE AND VIOLENCE TWO SYMPTOMS OF THE SAME PROBLEM BEHAVIOUR SYNDROME?

Despite the wealth of information that shows an association between alcohol use and violence in cross-sectional studies, questions remains as to whether the association is directly causal, or whether there may be a developmental association, that is, that both behaviours develop from common underlying factors. It is well known that problem behaviours cluster within individuals; for example smoking in adolescence is associated with delinquent behaviours such as property crime and poor school achievement (Ellickson and McGuigan, 2000), and substance use, antisocial behaviour and physical inactivity have been found to cluster together (MacArthur *et al.*, 2012). It is therefore possible that both alcohol misuse and violence cluster together due to a common underlying factor that predisposes to both.

Alcohol misuse and drug misuse have been conceptualized as problem behaviours similar to delinquency (Zucker, 2006). An understanding of the aetiology of problem behaviour in general can therefore be applied to the aetiology of problem drinking and substance misuse. A general deviance framework, such as the Problem Behaviour Theory (Jessor and Jessor, 1977), suggests that the extent to which individuals engage in problem behaviours is due to their innate general propensity to engage in problem behaviour; according to the theory this general factor is key in determining the expression of problem behaviour. This general propensity is made up of combinations of motivations and beliefs about control

(DeCoutville, 1995), impulsivity (Gottfredson and Hirschi, 1990, Stacy and Newcomb, 1993), or neuropsychological deficits (Moffitt, 1993). Although this theory incorporates the importance of social and contextual factors, the core idea in this model is that the propensity is an innate and stable characteristic.

In accordance with this model of understanding, some studies have investigated whether there is statistical evidence for a common factor underpinning substance misuse and antisocial behaviour or violence by attempting to explain the patterns of covariation between the two using confirmatory factor analysis. These studies, however, have tended to show that a single common factor cannot adequately explain both alcohol use and violence (at least statistically). For example, a prospective study of individuals aged 12, 15, and 18 over 3 waves used principal component analysis to analyse whether variation in substance misuse and delinquency could be accounted for by a single common factor (White and Labouvie, 1994). They found that delinquency and substance misuse represented two different dimensions of problem behaviour. Another study employed factor analysis to analyse data from a survey of 194 13-18 year old homeless people carried out every 3 months for 15 months. The authors found that substance use and delinquency did not coalesce statistically, and a 2-factor solution better fitted the data (Paradise and Cauce, 2003).

Some authors have suggested that although there may be a common underlying factor that broadly contributes to a tendency towards deviance, there are also specific factors that influence behaviours. Osgood and colleagues (Osgood *et al.*, 1988), for example, separated deviant behaviour into several components, which included intentional victimisation of other people, heavy alcohol use, cannabis use, and other drug use and found that both general tendency towards deviance (such as personality) and specific factors (such as substance use) were important. A similar approach used by Dembo and colleagues (Dembo *et al.*, 1992) was an attempt to replicate the earlier Osgood findings. They found that several problem behaviours represented, in part, a manifestation of a general tendency towards deviance and, in part, a unique phenomenon. The exception was alcohol use in which the variance across time was accounted for by the general tendency towards deviance, and not due to any additional alcohol-specific factor. A longitudinal study of antisocial behaviour (rather than violence) and alcohol use in adolescence showed that neither behaviour predicted the other between the ages of 12 and 13, but antisocial behaviour predicted alcohol use between ages 13 and 17, and among

males, alcohol use predicted antisocial behaviour between age 15 and 17 (Cho *et al.*, 2014). The change in the relationship between the two over time indicates that both behaviours can not simply be explained by a single underlying factor.

In summary, these studies do not appear to support the notion that substance misuse and violence are simply different expressions of the same underlying propensity, however they are limited having analysed data collected cross-sectionally.

LONGITUDINAL RELATIONSHIP BETWEEN ALCOHOL AND VIOLENCE

Studies with a longitudinal design enable the temporal sequence between an exposure and outcome to be investigated and can therefore address questions as to causation and reverse causality. The measurement of and statistical adjustment for other variables can address questions as to whether, and to what extent, the observed relationship can be explained by confounding.

In this section I summarise important longitudinal studies that have investigated alcohol use and violence. Studies were selected using methodology described by Farrington (Farrington, 2014) in which preference was given to studies that had as many of the following criteria as possible:

A prospectively chosen, general population sample (as opposed to retrospective comparisons);

A longitudinal design spanning at least 5 years (which makes it possible to establish casual order, to study the strength of effects at different ages, and to control extraneous variables better by investigating changes within individuals);

A large sample size of at least several hundred;

A large number of different types of variables measured (which makes it possible to study the effect of one independently of others, or interactive effects).

As can be seen from Table 1 (page 21) and Table 2 (page 33) there is conflicting evidence. I will present a summary of the evidence in favour of a conclusion that alcohol is associated with later violence, and a summary of the evidence that that

alcohol may not be related to related to later violence, before discussing the possible reasons for the discrepancy.

EVIDENCE THAT ALCOHOL IS ASSOCIATED WITH LATER VIOLENCE

The temporal sequence of minor delinquency, alcohol use, cannabis use and serious violence was investigated in the National Youth Survey Family Study (Elliott, 1994). This study investigated the order in which these behaviours occurred. They found that among those that had started drinking alcohol, first alcohol consumption preceded first serious violence in 86% of cases. Although this was a prospective longitudinal study, the analyses were designed only to investigate the proportion in which the onset of one behaviour preceded the other did not attempt to analyse possible causality by analysing the association between exposure and outcome.

A different approach, latent growth modelling, was carried out on data from 480 13-year-olds assessed annually over 4 years and then again 5 years later (Duncan *et al.*, 1997). This study aimed to investigate whether alcohol use reported at baseline was associated with later aggression, or whether change in alcohol use over the course of the study was associated with subsequent violence. The authors found evidence of an association between the amount of alcohol consumed at baseline and later levels of aggression in both males and females. In addition the rate of increase of drinking was also associated with levels of aggression, but only in males. However, the level of aggression at baseline was not adjusted for, and therefore it is possible that the more aggressive individuals were already drinking more. In addition, the sample was not nationally representative, was relatively small, and there was no adjustment for potential confounders.

Two studies arising from analysis of data from the Pittsburgh Youth Study have pointed to differing conclusions. The Pittsburgh Youth Study is a longitudinal study of 1,517 boys aged 7, 10 or 13 who were interviewed and then followed every 6 months for 3 years then every year for 9 years. White and colleagues analysed data from the older group, comprising 506 boys aged 13 in 6 waves of annual data collection up to age18 (White *et al.*, 1999). Violence was defined as a combined total of 6 acts which included carrying a hidden weapon, "strong arming" and rape or attempted rape. Using this definition of violence, the prevalence of violence, unlike in most studies of violence, tended to increase with age. After

controlling for possible common risk factors, including property crime and hyperactivity/impulsivity/attention problems, they found a significant longitudinal association between alcohol at baseline (age 13), and violence occurring at any time between age 14 and 18. However in their series of 5 analyses, which investigated the relationship between alcohol use in one year and violence in the next, the relationship was significant in only one consecutive year pairing (age 13 alcohol and age 14 violence). It is therefore not clear whether there is a relationship between alcohol and violence only during the earliest teenage years, or whether there may have been insufficient power to detect a relationship in subsequent years. It is also notable that longitudinal methods to deal adequately with repeated measures are relatively new, and this paper preceded such methodology. In a subsequent analysis of the younger group of boys who participated in the Pittsburgh Youth Study, 503 boys aged 7 followed up over 6 waves until age 20, no significant relationship was found between drinking alcohol and later violence (see also below).

Several papers have emerged from the study of a birth cohort in Christchurch, New Zealand. This is a prospective cohort study of 1,265 people over a period of 30 years. An analysis of these data covering the period when individuals were age 15-21 was carried out using both fixed-effects models that control for unmeasured time invariant confounders, and conventional regression methods controlling for measured fixed and time-varying covariates to try to ascertain whether there remains a significant relationship between alcohol and violence even after both measured and unmeasured factors are adjusted for. Both methods returned similar results, and found that there remained a significant relationship between the two (Fergusson and Horwood, 2000). The analyses were further extended to cover individuals up to age 30, again using fixed-effects models with time-varying covariates. For both males and females, the conclusions were the same. There was a significant association between the number of alcohol abuse or dependence symptoms and violence. Those with one or two alcohol abuse /dependence symptoms had one and a half times the rate of violence than those with none, and those who had five or more alcohol abuse / dependence symptoms had rates of violence three and a half times higher than those with no symptoms(Boden *et al.*, 2012).

Another analysis of data from the Christchurch Health and Development Study (Wells *et al.*, 2004) using latent class analysis attempted to differentiate 16-year-

olds on the basis of severity of alcohol use and misuse. The authors identified 4 latent classes, however it appeared that each of the classes represented increasing levels of severity of alcohol use, and were therefore likely to represent arbitrary categories on a spectrum of severity. They found that severity of alcohol problems predicted violence at age 16-21 but not age 21-25. The linear association between alcohol use (quantity, frequency, problems caused as categorised by latent class) and violence remained significant after controlling for relevant background factors.

In a longitudinal study of 6,338 children in California and Oregon, (Ellickson *et al.*, 2003) evidence that early drinking was associated with later violence was found. Compared to non-drinkers at age 12/13, drinkers were around 1.5 times more likely to be violent by age 17/18, and at age 23, twice as likely to engage in, "stealing, felonies or predatory violence"; these associations were not, however, adjusted for potential confounders including problem behaviour / delinquency at grade 7.

Two papers by Swahn and Donnovan used Add Health data to investigate relationships between alcohol and violence. The first paper (Swahn and Donovan, 2004), using waves I and II from Add Health investigated the correlates of new onset (incident) violence between the two time points. The first paper was an analysis of only the subgroup of adolescents who were drinkers at baseline. The study was therefore not designed to investigate whether drinking alcohol is associated with later violence, but did investigate, amongst others things, whether the quantity and frequency of drinking alcohol was associated with violence, and with the initiation of violence. They found that initiation of violence among the drinkers was associated with high volume or frequent alcohol use, low school achievement and expulsion and illicit drug use (see Table 1). Another limitation of this study in terms of investigating violence was that the measure included forms of antisocial behaviour such as whether the individual robbed someone, whether or not physical violence had taken place. In the second paper (Swahn and Donovan, 2005) the authors investigated predictors of incident alcohol-related fighting among 5,230 current drinkers between wave I and II, and investigated a larger number of possible predictors. They found that frequent drinking, and high volume drinking, (as well as factors such as, low college expectations and participation in weekly sports) were significantly associated with new onset alcohol-related violence. The problem with using alcohol-related fighting as an outcome measure is that it relies on the self-reported perception that alcohol caused the fighting, that

it does not necessarily involve serious violence, and would exclude other violence that the individual does not attribute to drinking alcohol. The authors recommended programmes to reduce frequent high volume drinking as a means of reducing violence. Assuming the findings support causality, it would be reasonable to expect that reducing drinking would reduce violence; however because sports participation and low college expectations were also associated with violence, the data would also support a recommendation of a reduction in weekly sports participation or strategies to increase college expectations as equally effective strategies.

Investigation of 2,335 13-year old students in Minnesota found that those who used alcohol at baseline were more likely to be violent 18 months later than those who did not (event rate ratio 1 1.2-1.7). With regard to binge drinking, girls who engaged in binge drinking had a significantly lower rate of violence than girls who never binged (rate ratio 0.41-0.95)(Blitstein *et al.*, 2005). The sample was not nationally representative as it was drawn from schools in low-income neighbourhoods, and the measure of violence included carrying a weapon (whether or not it was used in a threatening or violent manner). In addition, the study did not account for alcohol use in the intervening time between baseline and follow-up.

The 1970 British Birth Cohort Study is a study that surveyed eleven and a half thousand people at age 16, and surveyed again at age 30. The study showed that among 4,911 for whom outcome data was available, those who binge drank at age 16 had twice the odds of having any type of conviction by age 30 (Viner and Taylor, 2007), although there was no analysis of convictions specifically for violent offences. This study, although from a general population cohort suffered a very high attrition rate (70%). The analysis of convictions did not specify if they were convictions for violent offences, and there was no measure of alcohol use other than at baseline.

A study of 649 14-year old African American children who were considered to be at risk of dropping out of school were prospectively followed up annually for 4 years, then again after another 4 years. Xue and colleagues (Xue *et al.*, 2009) investigated trajectories of alcohol use and violence while controlling for other potential risk factors, including depression, academic achievement, selling drugs, parental violence, parental drug use, family conflict, gender and socio-economic status.

Using growth-curve analysis they investigated whether alcohol use affected the trajectory of violence, and whether violence affected the trajectory of alcohol use. They found that early alcohol use predicted later violence, and vice versa. They also found that change in one behaviour was associated with change in the other, leading the authors to conclude that there is a bi-direction relationship between alcohol and violence. This study, like the Christchurch Health and Development studies was one of the few that incorporated analysis of changes in alcohol use and violence at each data collection point. The study appeared to show that the relationship between alcohol and violence was stronger in adolescence than in adulthood. This study however was based on a highly selective sample.

Another study from a highly selected sample came from data collected prospectively form 517 community psychiatric patients who had a history of heavy substance misuse or violence (Mulvey *et al.*, 2006). This study was unique however in the frequency and short amount of time between data collection points; patients were interviewed weekly over 26 weeks about their alcohol consumption and violence. The study investigated whether alcohol on one day predicted violence the following day. Although no potential confounders were adjusted for, the study found that violence was more likely to occur when alcohol had been consumed on the previous day (OR 1.8-3.2).

A study of 3,038 12-year-olds reassessed annually on two occasions investigated trajectories of violence and examined the extent to which alcohol use was associated with different trajectories. There were 4 categories found which described the trajectories of violence which the authors labelled "*escalators*", *chronic aggressive*", "*desistors*", and "*non-aggressive*". They investigated the extent to which baseline alcohol use was associated with each of the 4 categories of trajectories of violence. They found that those who were drinking alcohol at age 12 were twice as likely to be either chronic aggressive, or escalators after controlling for other baseline variables. This study however only included alcohol at baseline in the analysis, not at subsequent time points, and there were a limited number of confounders controlled for.

The Woodlawn study selected 6-year-old urban African American pupils from Chicago for participation in a longitudinal study, that followed them (so far) to the age of 42. The association between self-reported lifetime frequency of drinking at the age of 16 and adult violence (measured by official arrest records and self-report

at age 32 and 42) was investigated. There was a high attrition rate (approximately 40% of the participants). The authors found that adolescents who were frequent drinkers were around 1.7 times to be arrested in adulthood for a violent offence.

The final study (Scholes-Balog *et al.*, 2013) included 849 adolescents randomly selected from schools in Australia, and who were followed up from age 13 to age 15. Two measures of alcohol use were used, which were the frequency of drinking, and frequency of binge drinking (5 or more drinks in a row). A cross-lagged model was used to investigate the extent to which either frequency of drinking or frequency of binge drinking predicted later violence and vice versa. Alcohol consumption at age 13 was found to be associated with violence 2 years later after controlling for confounders (alcohol consumption at age 15 was not associated with violence at age 17). There was some evidence that violence at age 15 was associated with binge drinking at age 17. This study is one of the few to have adjusted for time-varying covariates, however the selection of covariates was limited, and did not include co-existing delinquent or antisocial behaviour.

Table 1. Summary of longitudinal studies showing that alcohol is associated with later violence

Name and description	Description	Violence measure	Alcohol Measure	Variables adjusted for	Findings	Comments
The National Youth Survey (Elliott, 1994) Country: USA	1,725 11-17 year olds interviewed over 8 annual waves.	Serious violent offences (aggravated assault, robbery, rape that involved injury or use of weapon).	Not reported.	None.	Among those who were violent and reported drinking alcohol, the initiation of alcohol consumption preceded the initiation of serious violence in 86%.	1. Study designed to investigate the temporal sequence of initiation of alcohol and violence among those who engage in both. Unable to address questions of causality.
Longitudinal study on predictors and consequences of substance use (Duncan *et al.*, 1997) Country: USA	763 Adolescents aged 11-15 from 2 North-Western urban areas in USA. Assessed annually for 4 years and then 5-6 years later. Analysis by latent growth modeling.	Frequency of aggressive behaviours in last 12 months e.g. involvement in fist fights or gang fights	5-point scale – self report frequency of drinking.	None reported.	Baseline and rate of increase of alcohol use at age 13 was correlated with later aggression in males (0.11 and 0.21) , but only baseline alcohol in females (0.13).	1. Not a nationally representative sample. 2. Analysis not on full sample n=480 (39% attrition). 3. No adjustment for potential confounders. 4. Baseline violence not adjusted for in analysis. 5. Causality can not be deduced.
Pittsburg Youth Study (PYS) (White *et al.*, 1999) Country: USA	1517 boys age 7, 10 or 13 followed every 6 months for 3 years then every year for 9 years. analysed 506 boys in 6 waves of annual data from age 13-18.	Any of 6 violent behaviours (carried a hidden weapon, strong arming, attacking with weapon with intent to seriously hurt or kill, hurt / threatening to hurt / forced or attempting to force someone to have sex.	Frequency of alcohol use.	Property crime Sexual intercourse frequency Academic achievement Depressed mood ADHD symptoms Poor communication with parents.	Alcohol use was associated with violence the next year and vice versa. Attention/impulsivity only control variable that was associated with later violence. Change in violence was associated with change in alcohol and vice versa.	1. Only sub-sample of those recruited were included in analyses (506 boys out of 1517 boys and girls). 2. Analysis not using specific longitudinal methods (series of binary comparisons from one year to the next using dichotomized variables. 3. Limited number of variables adjusted for.
Christchurch Health and Development Study (Fergusson and Horwood, 2000)	All 1365 children born in Christchurch in mid 1977. Studied at birth, 4 months, 1 year, annually to age 16, then	Self-report Delinquency Inventory + questions on total number of offences.	Age 16 – frequency and quantity, and alcohol related problems.	Gender Age of mother Family structure Maternal education SES	Significant association between alcohol and violent crime after controlling for confounders. For every	1. Good general population cohort study 2. Limitation of the regression models reported is that they produce only an overall effect

Country: New Zealand	at age 18, 21, 25 and 30. Fixed-effects regression analysis of data up to age 21	Derived measure of total number of violent offences		Family living standards Parental change Parental violence Physical punishment Sexual abuse Parental alcohol consumption Parental depression/anxiety Parental drug use Parental offending Conduct problems / delinquency Property offences IQ Attention problems Neuroticism Self esteem Parent attachment Novelty seeking Deviant peer associations Cigarette smoking Cannabis use Depression / anxiety Early sexual activity	increase in alcohol abuse symptoms, the rate of violent crime increased by a factor of between 1.1 and 1.2.	size and therefore do not estimate the extent to which the estimated effect varies by age.
Christchurch Health and Development Study (Wells *et al*., 2004)	All 1365 children born in Christchurch in mid 1977. Studied at birth, 4 months, 1 year, annually to age 16, then at age 18, 21, 25 and 30. Latent class analysis. 963 included in analysis. Related patterns of drinking at age 16 to outcomes at age 16-21 and 21-25.	Self-report Delinquency Inventory (number of incidents of gang fighting, strong-arming, attacking with weapon or intent to seriously hurt or kill, rape or forced sex).	Number of times drank alcohol, amount of alcohol consumed, largest amount consumed on a single occasion, at age 16.	Hard drug use Property crime Low academic achievement Depressed mood	4 latent classes identified which represented a single dimension of severity. Severity of drinking at age 16 was correlated with violent offending ages 16-21 and 21-25.	1. Good general population cohort study. 2. Analysis investigated whether there was a linear trend between latent classes of alcohol use at age 16 and later offending therefore loses information at the individual level in analysis. 3. Few potential confounders controlled for.
Christchurch Health and	All 1365 children born in	Self reported	Self-reported DSM-	Fixed effects model +	Significant association	1. Good general population

Development Study (Boden *et al*., 2012) Country: New Zealand	Christchurch in mid 1977. Studied at birth, 4 months, 1 year, annually to age 16, then at age 18, 21, 25 and 30. Fixed-effects model of association between alcohol misuse symptoms and violence over 30 years follow-up.	violence and intimate partner violence.	IV alcohol abuse symptoms.	time-dynamic factors : History of anxiety disorder History of depressive disorder Stressful life events (feeling bad scale) Cannabis or other drug use Unemployment Peer/partner substance use Peer/partner or offending	between the number of alcohol abuse or dependence symptoms and violence after controlling for confounders. Incident rate ratio of violence of between 1.9 and 3.6 among those with 5 or more symptoms of alcohol abuse.	cohort study. 2. Limitation of the regression models reported is that they produce only an overall effect size and therefore do not estimate the extent to which the estimated effect varies by age.
High risk community psychiatric patients in Northeastern USA (Mulvey *et al*., 2006) Country: USA	Prospective study of 517 attendees of emergency room of psychiatric hospital in USA who had a recent history of heavy alcohol or drug use and violence. Followed up of 26 weekly interviews. Investigation of whether alcohol predicted violence the following day or vice versa.	Number of drinks per day.	Number of incidents of serious violence (physical injury, threat with weapon, use of weapon, sexual assault).	None reported.	Alcohol use significantly predicted violence the following day, but violence did not predict alcohol use the following day.	1. Not a general population survey. 2. No potential confounders controlled for.
Seattle Social Development Project, (Huang *et al*., 2001) Country: USA	808 students age 10 in 18 schools in Seattle. Assessed annually to age 16 then every 2-3 years to age 27. Used SEM to investigate cross-lagged relationship between alcohol use and aggression over 4 waves.	Number of self-reported criminal activities at age 21 (not specifically violence). Number of self-reported criminal activities in the last year assessed at age 21 and 24 How often in past year: thrown rocks at people, picked a	Binge drinking (5 or more drinks in a row). Frequency of drinking alcohol (3 point scale) measured annually from age 14 to 18.	Gender Teacher reported internalizing behavior (anxiety, withdrawn) Teacher reported inattention / hyperactivity Property crime Academic achievement Parental supervision Parental attachment Neighbourhood desirability	After controlling for common risk factors measured at age 10, only one path, alcohol use at age 16 was significantly associated with aggression at age 18. Aggression was not associated with later alcohol use.	1. Sample selected from schools in high crime neighbourhoods. 2. Conflicting evidence of association – significant association found in only one out of three paths tested. 3. No time-dynamic factors controlled for.

		fight, hit people with the intention of hurting them.				
California and Oregon Longitudinal Survey (Ellickson *et al.*, 2003) Country: USA	6,338 children from school in California and Oregon assessed at age 12-13, and again at age 23	Age 17-18 and age 23 - items for "predatory violence" and "relational violence".	Number of times drank in last year (classified as non-drinkers, experimenters and drinkers). At age 23 3-item lifetime alcohol disorder screen and 5-items from Drug Abuse Screening Test	Cigarette smoking Cannabis use Other illicit substances School and employment problems Stealing Early pregnancy / parenthood Drug selling	Compared to non-drinkers at grade 7, Drinkers at grade 7 were 1.3-1.8 times more likely to engage in violence at grade 12, and at age 23 twice as likely to engage in predatory violence.	1. Limited longitudinal methodology – two separate analysis of baseline drinking and problem behaviours at two subsequent time points. 2. Not clear whether baseline violence was adjusted for in analysis.
Add Health (Swahn and Donovan, 2004) Country: USA	Analysis of sample of 8885 drinkers age 12-21 to investigate correlates of initiation of violence 1 year later.	Any of 6 violent behaviours	Drinking frequency Usual drinking quantity Problem drinking High volume drinking	Family structure Mother's education Shared decision making Relationship with parents Family activities Depression History of counseling Self esteem Peer alcohol use Parental alcohol use Drug use Exposure to drug use Delinquency School functioning Activities Alcohol consumption	Drinking 7 or more drinks each occasion was associated with later violence (OR between 1.2 and 1.7). Initiation of violence among drinkers was marginally associated with high-volume drinking (OR 1 – 2) in a model that did not include interaction terms, but was not significant when interaction terms were included in the model.	1. Analysis only on subsample of adolescents who were drinkers. 2. Measure of violence included items in which not physical violence may not have taken place (robbery).
Add Health (Swahn and Donovan, 2005) Country: USA	Longitudinal analysis of current drinkers age 12-21 to investigate correlates of alcohol related violence 1 year later (n=2,990).	Fighting attributed to alcohol use (dichotomised) or a "serious physical fight".	Drinking frequency Usual drinking quantity Problem drinking	Family structure Mother's education Shared decision making Relationship with parents Family activities Depression History of counseling	High-volume drinking, males, drinking >9days per month, trouble in school, low college expectation and sport activities associated with drinking-related fighting in multivariate logistic model.	1. Analysis only on subsample of adolescents who were drinkers. 2. Violence measure was initiation of self-reported alcohol-related fighting, therefore relies on participants attributing

				Self esteem Peer alcohol use Parental alcohol use Drug use Exposure to drug use Delinquency School functioning Activities Alcohol consumption	Initiation of alcohol related violence at wave II associated with alcohol use, drug use and selling, exposure to drugs, delinquency and poor school functioning.	violence to alcohol and excludes other forms of violence.
TEENS study, Minneapolis (Blitstein *et al*., 2005) Country: USA	2355 students in Minneapolis age 13 followed up 18-months later.	How many times in last year engaged in one of 5 violent behaviours (carry a weapon, hit or beat up someone, group fighting, hurt someone badly enough to need bandages or a doctor, use knife or gun to get something from someone).	How many times in the last 30 days have drank alcohol. How many times in last 2 weeks engaged in binge drinking (5 or more drinks in a row).	Ethnicity Gender Depressive symptoms Influence of spirituality Future onlook Parenting style Cigarette smoking Illicit drug use	Alcohol users 1.2-1.7 times more likely to engage in violence. Male binge drinkers no more likely than non-binge drinker to be violent. Binge drinking girls less likely to be violent.	1. Not a general population study (sampled from low income population). 2. Main purpose of analysis was to determine if predictors of violence differed between boys and girls. 3. Alcohol use other than at baseline was not measured.
1970 British Birth Cohort (Viner and Taylor, 2007) Country: UK	16567 Infants born in UK between 5th and 11th April 1970. Followed up at age 5, 10, 16 and 30 years. Analysis of 4911 at age 30 who had data available at age 16	Formally cautioned by the police age 10-16, and Court convictions since age 16.	Age 16: Binge drinking (2 or more occasions of drinking 4 or more drinks in a row in the last 2 weeks). Frequent drinking (drinking on 2 or more occasions a week in previous year). Age 30 – alcohol quantity and CAGE questionnaire.	Ethnicity Income Occupation Illicit drug use Significant accidents Homelessness School exclusion	Binge drinkers at 16 twice as likely (OR 1.2-2.5) to be subsequently convicted by age 30 than non binge drinkers after adjusting for socioeconomic status and baseline police cautions.	1. Large general population survey, but high attrition rate (70%). 2. Outcome measure was convictions which were not necessarily for violent offences 3. No measure of alcohol use other than at baseline.
2004 Youth Violence Survey (Swahn *et al*., 2008)	4,131 students age 12, 14 and 17-18 in a high-risk community. Sample of 856 12-13	Scales for dating violence perpetration and peer violence	Age at first alcohol use. Analysed binary variables – alcohol initiation	Peer delinquency Parental monitoring Impulsivity Gender	Drinkers associated with peer and dating violence. Not significant after controlling for peer	

Country: USA	grade children included in analysis.	perpetration – created binary variable for each scale.	before age 13 and no use of alcohol.	Binge drinking Illicit substances.	delinquency and parental monitoring.	
Longitudinal study of youth at risk of dropping out of school. (Xue *et al*., 2009) Country: USA	681 14-year old African Americans in Michigan at risk of dropping out of school (low educational attainment). Assessed annually for 4 years, then once again 4 years later. Analysis of growth curves.	Frequency of alcohol use over previous 12 months	Number of times engaged in one of 7 items of violent behaviours (fought in school, engaged in a group fight, hurt someone to need bandages or a doctor, hit a teacher or supervisor, used a weapon to get something, carried a knife, carried a gun).	Depression Academic achievement Selling drugs Peers violent behavior and drug problems Parental violent behavior and drug problems Family conflict Gender Socioeconomic status	Violence peaked in middle/late adolescence and declined, but alcohol use increased. Early alcohol predicted later violence and vice versa. Changes in one behaviour associated with changes in the other.	1. Not a nationally representative sample. 2. One of the few studies to have investigated longitudinal trajectories.
Project Northland Chicago (Maldonado-Molina *et al*., 2010) Country: USA	3038 urban youths age 12, followed up at age 13 and 14.	Frequency of drinking alcohol over the past 12 months.	4 items, Told someone you were going to hit or beat them, pushed, shoved or pulled hair, kicked, hit or beat someone, or group fighting.	Age Ethnicity Family structure Nationality Trouble with the police Time supervised by a adult Peer delinquency	Latent class analysis of trajectories of violence found 4 classes, “non aggressive” “Escalators”, “Chronic aggressive” and “desistiors”. Found that alcohol use were twice as likely to be either in escalators or chronic aggressive group after controlling for baseline characteristics.	1. Only baseline alcohol use was used in analysis. 2. Limited adjustment for potential confounders.
Woodlawn Study Country: USA	702 African Americans followed from age 6 to 42	Lifetime frequency of alcohol consumption at age 16	Arrests for violent offences (from age 17) and self-reported violence at age 32 and 42	Propensity score matched on: Gender Socio-economic status Family background School achievement School adaptation Delinquency Smoking Mother’s education	Logistic regression following propensity score matching. Frequent adolescent drinkers twice as likely to be arrested for assault in adulthood compared with light/non-drinkers. Effect was mediated by binge-drinking in adulthood.	1. Not a nationally representative sample. 2. High attrition rate (40.3%). 3. One of few studies to include official records, and self-report.

				Family mobility Family discipline Family substance use 1st Grade teacher ratings		
International Youth Development Study Country: Australia	849 youths age 13, assessed on 2 further occasions at age 15 and 17.	Frequency of consuming alcohol, and frequency of drinking 5 or more drinks in a row.	How many times beaten someone so badly they needed to see a doctor or nurse, and frequency of attacking someone with the idea of seriously hurting them.	Academic failure Time dynamic: Friends drug use Antisocial friends Family conflict Depressive symptoms Time invariant: Parent education level Early antisocial behavior	Cross-lagged SEM model Small association between alcohol consumption at age 13 and violence at age 15 after controlling for confounders. Association between binge drinking at 15 and violence at 17 after controlling for confounders	1. One of the few studies that adjusted for time-varying confounders. 2. Did not control for concurrent delinquency or antisocial behavior.

EVIDENCE THAT ALCOHOL MAY NOT BE ASSOCIATED WITH LATER VIOLENCE

Having summarised the main studies that have shown evidence of an association between alcohol and violence, I will now summarise studies that have shown only weak or no evidence of an association. As stated above, one of the studies arising from the Pittsburgh Youth Study investigated the relationship between early alcohol use and later violence over a 12-year period (Wei *et al.*, 2004) and found little convincing evidence of a relationship in either direction. Eight sets of calculations were carried out to test whether alcohol use one year was associated with violence the following year, while controlling for the effect of violence and cannabis use at baseline. The study found that frequent alcohol use at age 11 was associated with violence at age 12 (OR 2.7), but after age 11 there was no significant relationship between alcohol use one year and violence the next. This is in contrast to the earlier paper from the Pittsburgh Youth Study (White *et al.*, 1999) described above. The authors suggest that the discrepancy may be because different cohorts of children within the study were included in each paper (in the earlier paper the youngest in the cohort of children were selected for analysis, and in the subsequent paper, and older children were selected) and therefore a "cohort effect" may be present. Another possibility suggested by the authors is that differences may be due to the different age range used for measurement for both the exposure and the outcome in both studies (the first study examined behaviours at age 13 predicting outcomes at age 14-18, and the second examined behaviours at age 11-14 to predict behaviours at age 15-20).

A very small study of 136 12-18 year old males using structural equation models, found that early aggressive behaviour was associated with later alcohol use, but levels of alcohol abuse was not related to later levels of aggression (White *et al.*, 1993). Criticisms of this study are that the sample size is small that the measure of aggression was diverse and included vandalism, and the only potential confounding variable for which the authors controlled for was parental education level.

Using latent class analysis of data from 808 10-year old and children followed them prospectively until age 21. They investigated patterns of binge drinking and identified 4 categories which they named "*non binger*", "*increaser*" "*high early*" and "*late onsetter*") (Hill *et al.*, 2000). Analysis found no relationship between the category of binge drinking and crime measured at age 21. It should be noted that in

this study, the outcome measure included non-violent crimes, and only included crimes reported in the 12 months preceding the final interview, and therefore did not take into account any other crimes committed over the preceding 10 years of the study.

A longitudinal study in the UK investigated alcohol use and antisocial behaviour of 2,586 11 year olds and followed them again at 13 and 15 (Young *et al.*, 2008). Also using structural equation models, the authors found that antisocial behaviour led to later alcohol abuse, but found no evidence that alcohol abuse led to antisocial behaviour. Of note, the measure used was antisocial behaviour rather than violence, and in addition the was only a very limited number of variables controlled for.

Another study which involved structural equation analysis of over 808 students over 4 waves found only weak evidence of a relationship between alcohol and aggression in one out of the three paths tested (Huang *et al.*, 2001).

A longitudinal study from the Seattle Social Development Project investigated the rate of change in alcohol use among 808 youths selected from high-crime neighbourhoods, assessed annually from age 14-18 as to whether alcohol was associated with crime at age 21 and 24. The measure of crime was a self-report measure of the number of criminal activities (non-specifically violent acts) within the past year. The analyses used latent growth curve modelling to estimate a baseline (termed the intercept), and rate of change (the slope) in drinking, and then examined whether either the baseline or rate of change was associated with crime. Although early delinquency was associated with both crime and alcohol use, there was no association between alcohol use and crime (either baseline or rate of change) once delinquency was controlled for (Mason *et al.*, 2010).

A large study of 4,131 students selected individuals from a high-risk community analysed retrospective data relating to the age of initiation of alcohol use on the rate of subsequent violence (Swahn *et al.*, 2008). The study found no relationship between age of alcohol initiation and either later peer or dating violence once peer delinquency and parental monitoring were controlled for. This study however was not able to investigate whether subsequent or current alcohol use was associated with violence.

Add Health data was used to categorise participants at baseline in one of four categories based on the presence or absence of both alcohol use and violence. Two sets of analysis were carried out to investigate whether any of the 4 categories were associated with violence at the final data collection point (Reingle *et al.*, 2011). They found that (in unadjusted analyses) those who were violent at baseline, whether or not they used alcohol were more likely to be violent at the endpoint (OR 1.1-2.8), but those who were non-violent, whether or not they used alcohol at baseline were not more likely to be violent at the end of the study. Results were not presented for the full sample after adjusting for potential confounders, but only by race as this was the primary focus of the study. This study adjusted for very few potential confounders and did not take into account any dynamic factors, especially changes in alcohol use during the course of the study. A related study by the same authors identified three trajectories of violence which they named "*nonviolent*", "*escalators*" and "*desistors*". The authors found no evidence that alcohol use at baseline was associated with any of the trajectories (Reingle *et al.*, 2012a).

POSSIBLE REASONS FOR DIFFERENCES IN FINDINGS

Differences in findings may be due to at least four possible reasons. First, as can be seen from Table 1 and Table 2, there are differences in the definitions of violence. Some studies have incorporated broader behaviours of antisocial behaviour such as vandalism in their definition (e.g. (Huang *et al.*, 2001, White *et al.*, 1993), whereas others have restricted the definition to clear acts of physical violence (e.g. (Wei *et al.*, 2004).

Second, many studies have failed to control for the breadth of potentially relevant factors. Several studies controlled for only three or fewer potential confounders (Duncan *et al.*, 1997, Fite *et al.*, 2007, Gruber *et al.*, 1996, Hill *et al.*, 2000, Mason and Windle, 2002, Mulvey *et al.*, 2006, White *et al.*, 1993, Young *et al.*, 2008), and of those, four of the studies had not controlled for any. Several studies used structural equation modelling as a method to investigate the relationships between the two (Fothergill and Ensminger, 2006, Huang *et al.*, 2001, Mason and Windle, 2002, White *et al.*, 1993, Young *et al.*, 2008). A variation of structural equation modelling is latent growth modelling, in which an estimate is made of the baseline level of the variable of interest and the rate of change; the extent to which other variables are associated with either baseline or rate of change can then be estimated (Dembo *et*

al., 2007, Duncan *et al.*, 1997, Hill *et al.*, 2000, Mason *et al.*, 2010, Wells *et al.*, 2004, Xue *et al.*, 2009). The advantage is that multiple relationships can be modelled simultaneously but the disadvantage is that fewer potential confounders are included in the models, most likely because with the addition of greater numbers of variables there is an increase in complexity of the models that can be understood diagrammatically (the main way that structural equation models tend to be presented in published studies), and a decrease in the likelihood that the models will converge and produce an estimate.

Third, the age of the participants both at inception and at follow up varies between the studies. Some studies recruited children aged 10 or younger (e.g. (Huang *et al.*, 2001, Mason *et al.*, 2010, White *et al.*, 1999), while others recruited older children and young adults. The age at which violence was measured as the outcome variable also varied considerably between studies, while some investigated overall trajectories of violence (e.g.(Maldonado-Molina *et al.*, 2010)), others investigated violence at fixed time points (e.g. (Huang *et al.*, 2001)). It is possible therefore that the relationship between alcohol and violence is not constant, and may vary with age, but this is unclear from the studies.

Fourth, few studies have incorporated changes in level of alcohol use during the course of the study, and have relied on baseline alcohol use and subsequent violence. It is difficult to draw conclusions about violence occurring in some cases several years after the measurement of alcohol use in the intervening time.

SUMMARY OF FINDINGS AND UNANSWERED QUESTIONS

In summary, although there have been numerous studies that have measured both alcohol and violence, very few have fulfilled all the criteria set out by Farrington above. Many studies have been carried out on highly selective groups, such as adolescents considered at high risk of offending, or the recruitment of individual specifically from schools or neighbourhoods where there is a high crime rate. Many studies also have been relatively small (less than 500 participants). Although violence has been the outcome measure used in many studies, there have been several studies that have investigated crime more broadly, or milder forms of aggressive or delinquent behaviour. Most studies have attempted to control for at least some potentially confounding variables, but few have included a

comprehensive set of variables that have been identified as being associated with both violence and alcohol use. Finally, a variety of statistical methods have been used with varying levels of sophistication, and not all have been optimal to the longitudinal design.

Having commented on the differences between the studies it is appears that:

There is strong evidence that individual who drink alcohol at a young age are also more likely to engage in other problem behaviours, including violence.

The association between alcohol and violence is markedly reduced after adjusting for confounding, and in some studies there is no association after adjusting for confounding. There remains equivocal evidence of whether alcohol is an independent risk factor for violence from prospective community studies.

High amounts of alcohol consumption including binge drinking appear to be associated more strongly with violence than low alcohol consumption. It is not clear whether drinking frequency, volume or both are equally important in the possible relationship with violence.

The relationship between alcohol and violence may change with age, hence the discrepancy in findings between studies, however it is not clear whether and to what extent this may be the case.

Table 2 Summary of longitudinal studies that show that alcohol may not be related to later violence

Name and description	Description	Violence measure	Alcohol Measure	Confounders adjusted for	Findings	Comments
Pittsburg Youth Study (PYS) (Wei *et al*., 2004) Country: USA	1517 boys age 7, 10 or 13 followed every 6 months for 3 years then every year for 9 years. Analysed 503 boys age 7 over 6 waves until age 20.	Any of 6 violent behaviours (carried a hidden weapon, strong arming, attacked with weapon with intent to seriously hurt or kill, hurt / threatened to hurt / forced or attempted to force someone to have sex.	Frequency of alcohol use.	Property crime Sexual intercourse frequency Academic achievement Depressed mood ADHD symptoms Poor communication with parents Hyperactivity / inattention / impulsivity Poor communication with parent Poor supervision Parent perception of bad neighbourhood Ethnicity	Frequent alcohol use at age 11 predicted violence at age 12, but no other ages (up to age 20). Aggregated data showed that drinking age 11-14 associated (OR 1.97) with violence age 15-20 after controlling for common factors.	1. Relatively small sample (503) 2. Included only boys. 3. Series of paired analyses rather than specific longitudinal methods.
Rutgers Health and Human Development Project. (White *et al*., 1993) Country: USA	431 adolescents age 12 interviewed again t age 15 and 18.	Hurting someone badly, using a weapon in a fight, vandalism, hitting parents, fighting in school.	Quantity of alcohol Frequency of alcohol, Largest amount consumed on one occasion, Number of times intoxicated in last year	Parental level of education.	Early aggressive behavior associated with increases in alcohol use, but levels of alcohol use not related to subsequent levels of aggression.	1. Small sample size 2. Few potential confounders adjusted for 3. Only 136 males included in final analysis
Seattle Social Development Project, USA (Hill *et al*., 2000) Country: USA	808 students age 10 in 18 schools in Seattle. Assessed annually to age 16 then every 2-3 years to age 27. Latent growth curve analysis of binge drinking trajectories.	Number of self-reported criminal activities at age 21 (not specifically violence). Number of self-reported criminal activities in the last year.	Binge drinking (5 or more drinks in a row). Frequency of drinking alcohol (3 point scale) measured annually from age 14 to 18. Frequency of binge-drinking (5 or more	Previous criminal behaviours Gender Socio-economic status Illicit substance abuse	Identified 4 trajectories (non-bingers, early highs, increasers and late onset). No association between trajectory and crime.	1. Outcome measure was all types of crime, not necessarily violence. 2. Outcome only measured crime over the preceding year at age 21. 3. Few control variables. 4. Analysis of whether identified latent class is associated with crime, rather than individual alcohol use.

			drinks in a row).			
West of Scotland 11-16 Study (Young *et al*., 2008)	2586 11 year old boys and girls, followed at age 13 and 15. SEM model of longitudinal relationship between antisocial behavior and alcohol use.	Latent variable for antisocial behaviour (miss school, trouble with police, take risks, get into fights, rule breaker). Latent variable for alcohol-related trouble (trouble with police, hurt self, fights, arguments, skipped school)	Latent variable created from alcohol frequency, ever been really drunk, length of time drinking (age commenced drinking).	Social class derived from job of head of the household Drinking context (drink provided by parents /others).	Antisocial behaviour led to alcohol abuse, but no evidence that alcohol abuse led to antisocial behaviour.	1. Measure used was antisocial behavior rather than violence, such as risk taking and missing school. 2. Very limited potential confounders analysed.
High risk community psychiatric patients in Northeastern USA (Mulvey *et al*., 2006) Country: USA	Prospective study of 517 attendees of emergency room of psychiatric hospital in USA who had a recent history of heavy alcohol or drug use and violence. Followed up of 26 weekly interviews.	Number of incidents of serious violence (physical injury, threat with weapon, use of weapon, sexual assault).	Number of drinks per day.	None reported.	Investigation of whether alcohol predicted violence the following day or vice versa. Alcohol use significantly predicted violence the following day, but violence did not predict alcohol use the following day.	
Youth Support Project (YSP) Hillsbourough County Juvenile Assessment Center (Dembo *et al*., 2007) Country: USA	278 youths arrested and processed at Hillsborough County Juvenile Assessment Center, followed over 4 years. Latent growth modeling of data.	Self reported delinquent behaviour	Self reported heavy drinking.	Cannabis use Psychological functioning (SCL-90) Family drug / alcohol use Family mental health problems Family criminality	Significant declining trend of delinquency over time. Alcohol and marijuana use slight increasing trend over time.	
2004 Youth Violence Survey (Swahn *et al*., 2008) Country: USA	4,131 students in grades 7, 9 and 11/12 in a high-risk community. Sample of 856 7th grade children.	Age at first alcohol use. Analysed binary variables – alcohol initiation before age 13 and no use of alcohol.	Scales for dating violence perpetration and peer violence perpetration – created binary variable for each scale.	Peer delinquency Parental monitoring Impulsivity Gender Binge drinking Illicit substances.	Drinkers associated with peer and dating violence. Not significant after controlling for peer delinquency and parental monitoring.	1. Not nationally representative sample 2. Investigated only the age of alcohol initiation and later violence
Project Northland Chicago (Maldonado-Molina *et al*., 2010)	3,038 urban youths age 12, followed up at age 13 and 14.	Frequency of drinking alcohol over the past 12 months.	4 items, Told someone you were going to hit or beat them, pushed,	Age Ethnicity Family structure Nationality	Latent class analysis of trajectories of violence found 4 classes, “non aggressive” “Escalators”, “Chronic	

Country: USA			shoved or pulled hair, kicked, hit or beat someone, or group fighting.	Trouble with the police Time supervised by a adult Peer delinquency	aggressive" and "desistiors". Found that alcohol use was associated with escalators and chronic aggressive group after controlling for baseline characteristics	
Seattle Social Development Project, USA (Mason *et al.*, 2010) Country: USA	808 students age 10 in 18 schools in Seattle. Assessed annually to age 16 then every 2-3 years to age 27. Latent growth curve modeling.	Frequency of alcohol in past month, quantity on a typical occasion, how often got drunk when drank alcohol.	Number of self-reported criminal activities in the last year assessed at age 21 and 24.	Gender Household income Early problem behaviours Early sexual activity	Neither baseline or rate of change of drinking was associated with later crime, but delinquency associated with later alcohol use.	1. Not a nationally representative sample - Participants selected from high-crime neighbourhoods. 2. Outcome measure was crime, not specifically violence.
Add Health (Reingle *et al.*, 2011) Country: USA	Nationally representative longitudinal study of 15000 adolescents in USA. Up to 4 waves Investigated effect of violence at wave I and II on alcohol use at Wave IV, and alcohol use at Wave I and II on violence at Wave IV using logistic regression.	3 or more DSM-IV alcohol abuse symptoms Usual drinking quantity Drinking frequency, high volume drinking unsupervised drinking problem drinking (drinking until intoxicated or drinking causes negative problems).	Self reported serious violence: "Pulled a knife or gun on someone" "Hurt someone so badly they needed bandages or a care from a doctor or nurse" "Shot or stabbed someone"	(Measured at wave I) Depression Academic achievement Parental involvement Perception that neighbourhood is safe Cannabis use Desire to leave home Peer alcohol and cannabis use Ethnicity Age Gender	Either alcohol, violence or a combination of the two predicted later alcohol abuse. Alcohol abuse did not predict later violence, unless history of early violence.	1. Did not account for changes in alcohol use during the study 2. Outcome was only violence at final data collection point 3. Few potential confounders adjusted for.
Add Health (Reingle *et al.*, 2012a) Country: USA	Nationally representative longitudinal study of 15000 adolescents in USA. Up to 4 waves Identified trajectories of violence and their correlates.	High volume drinking Unsupervised drinking Problem drinking (drinking until intoxicated or drinking causes negative problems)	Any violent behaviour out of a 6-item measure (dichotomised).	Depression Academic achievement Parental involvement Perception that neighbourhood is safe Cannabis use Desire to leave home Peer alcohol and cannabis use Ethnicity Age Gender	Trajectories identified" non-violent", "desistors", and "escalators". No association between alcohol use and "escalators", but association with desistors.	

CHAPTER 4 CANNABIS USE, CIGARETTE SMOKING AND VIOLENCE

EPIDEMIOLOGY OF CIGARETTE SMOKING AND CANNABIS USE IN THE GENERAL POPULATION

Cigarette smoking

In the UK, approximately 22% of adult men and 17% of adult women are tobacco smokers. The prevalence is similar in the USA, where around 21% of males and 16% of females smoke (Centre Health and Social Care Information, 2014). Smoking is strongly associated with lower socio-economic status, and rates in the UK vary from 60% in the most deprived to 15% in the most affluent people (Hiscock *et al.*, 2012). Around two-thirds of smokers start before the age of 18. Less than 0.5% of 11-year olds in England reported that they were regular smokers, rising to 8% of 15 year-olds (Health and Social Care Information Centre, 2014b).

Cannabis

Cannabis is the most commonly used illegal drug in the world; it has been estimated that around 3.5% of the world population aged between 15-64, corresponding to around 180 million people consumed cannabis at least once in the past year (United Nations, 2011). In England and Wales, 6.4% of adults aged 16-59 reported that they had used cannabis in the last year, and 30% reported lifetime use (Home Office, 2013). Some reports estimate the lifetime use of cannabis is up to 50% (European Monitoring Centre for Drugs and Drug Addiction, 2006, Perkonigg *et al.*, 2008) In North America, it is estimated that approximately 10.7% of the population use cannabis (United Nations, 2011).

EPIDEMIOLOGY OF TOBACCO SMOKING AND CANNABIS USE AMONG OFFENDERS

Cigarette smoking

Across Europe, tobacco smoking rates among prisoners are much higher than in the general population (at least twice as high) (Hartwig *et al.*, 2008), and in

England approximately 80% of prisoners are young offenders are smokers (Lader *et al.*, 2000, NHS Information Centre, 2011).

Cannabis

Evidence for association between offending and cannabis use comes from a variety of sources. Official data collected from arrestees and prisoners shows a high prevalence of cannabis use from among this population. The US Department of Justice reported data from the Arrestee Drug Abuse Monitoring program, which was set up to monitor the serum blood concentrations of drugs in arrestees. It reported that between 30 and 50% of arrestees tested positive for cannabis (Levinthal, 2008). Furthermore, it has been reported that around 80% of incarcerated males and females admitted to using cannabis at some point in their lives (Neff and Waite, 2007). Among prisoners in England and Wales, the prevalence is similar. Approximately 75% of male prisoners, and 70% of female prisoners reported ever having used cannabis, and around 60% of males prisoners and 45% of female prisoners reported having used cannabis in the year before they were incarcerated (Singleton *et al.*, 1999).

Cannabis users have been categorised as either "*experimental*", "*recreational*", or "*chronic*" depending on the pattern of use (United Nations, 2011). Chronic users have been differentiated from non-users and experimenters on the basis of individual characteristics; chronic users tend to score lower on measures of self-control, have lower aspirations, and show more externalising behaviour, and have a higher orientation to sensation seeking (Brook *et al.*, 2011). Experimental users are those who try using the drug during adolescence; they have poorer relationships with their parents, and tend to be novelty/sensation seeking. A longitudinal study of over 2000 adolescents in Australia found that heavy alcohol use and antisocial behaviour was associated with early cannabis use. Most users were occasional or intermittent users, but about 12% of the sample escalated to daily use by late adolescence. Daily cigarette smoking was a strong predictor of both initiation and persistence of cannabis use (Coffey *et al.*, 2000). Another longitudinal study of patterns of cannabis use included 540 students in Germany. Baseline factors that predicted cannabis use 10-years later were alcohol dependence, antisocial personality disorder, early onset cannabis use, peer drug use, young age at first use, and heavier cannabis use at baseline (Perkonigg *et al.*, 2008).

ASSOCIATION BETWEEN CIGARETTE SMOKING, CANNABIS AND VIOLENCE

Cigarette smoking

No studies were identified that specifically investigated the longitudinal relationship between cigarettes smoking and violence. Many of the longitudinal studies summarised in Table 1(page 21), Table 2 (page 33) and Table 3 (page 43) adjusted for cigarette smoking as a confounder, or combined it with either alcohol use or other drugs as a composite substance use variable to investigate the relationship with violence. Only one study (Blitstein *et al.*, 2005) reported separately the relationship between cigarette smoking and violence. In this study of 2,355 13-year old students from Minneapolis in the USA were asked how many times in the last 24 hours, and in the last 7 days had they smoked cigarettes. They were classified as either low/non smokers versus weekly or greater smokers. They were assessed again 18-months later and asked how many times in the past year had they engaged in serious violence. After controlling for several potential confounding variables, no association between cigarettes smoking and violence was found.

Laboratory animal studies suggest that the administration of nicotine reduces aggression. For example, laboratory studies of mice showed that there was a does-dependent reduction in attacking behaviour (Johnson *et al.*, 2003) and fighting (Driscoll and Baettig, 1981) following the administration of nicotine. Laboratory studies in humans have been carried out which measured aggressive responses (defined as the frequency of extracting money from an opponent in a research paradigm) following the administration of varying doses of nicotine. These studies also found a dose-dependent reduction in aggressive responses (Cherek, 1984). Most notably however, this study was carried out among smokers, and it is possible that nicotine was reducing irritability among smokers who were in a relative state of withdrawal. In addition, increased irritability has been observed among smokers during acute withdrawal of nicotine (Cherek *et al.*, 1991), with highest among those with high trait irritability (Parrott and Zeichner, 2001). The relationship between nicotine dependence and the severity of aggression was investigated among patients admitted for treatment for alcohol dependency in Turkey. It was found that smoking cigarettes was positively correlated with

aggression, although the authors suggested that the direction of causality may be in either direction (Saatcioglu and Erim, 2009).

Cannabis

Several studies have investigated the relationship between cannabis use and violence or delinquency, and the results have been mixed. There have been several studies that have shown that early aggressive behaviour is associated with the initiation of substances, including cannabis (e.g. (Fite *et al.*, 2007, 2008)) indicating that both may be part of a problem behaviour syndrome, however other studies have found no relationship (e.g. (Fothergill and Ensminger, 2006)). The extent to which cannabis is causally related to violence or aggression remains uncertain.

A systematic review and meta-analysis published in 1999 included 30 studies that reported associations between cannabis use and aggression (Derzon and Lipsey, 1999). This studied showed very modest correlations; the mean weighted correlation was 0.1. The effect size was the same for cannabis use preceding aggression, aggression preceding cannabis use, and aggression and cannabis use measured concurrently. It was also shown that the relationship was strongest in the younger age groups, and reduced with age. It appeared from this study that cannabis use and aggression are associated, but cannabis use does not in itself cause aggression. In contrast, a review of the literature on cannabis and interpersonal violence concluded that overall data supported an association between cannabis and violence, and theorised that cannabis withdrawal may be assosciated with violence (Moore and Stuart, 2005).

Several of the longitudinal studies described in Chapter 3 also reported an analysis of the relationship between cannabis use and later violence. In the National Youth Survey Family Study (Elliott, 1994) that investigated the temporal sequence of minor delinquency, alcohol use, cannabis use and serious violence, it was found that those who committed serious violence and had started cannabis use, cannabis use, preceded serious violence in 63% of cases. As stated in Chapter 3, causality cannot be deduced from this study.

The Pittsburgh Youth Study also reported the relationship between cannabis and violence (Wei *et al.*, 2004, White *et al.*, 1999, White *et al.*, 2002c). The studies showed a weak longitudinal association between cannabis use and later violence, which was not significant after controlling for common risk factors. They found

that those who started using cannabis at a young age were more likely to be deviant in other ways, including committing property offences, engaging in other drug use and violence. The authors concluded that cannabis use does not cause violence, but is associated with other common factors. The authors also compared frequency of fighting among alcohol and cannabis users, and found that those that drank alcohol were nearly 4 times more likely to get involved in fighting than those who used cannabis.

Blitstein and colleagues (Blitstein *et al.*, 2005) investigated 2,335 13-year old students in Minnesota investigating the relationship between cannabis use and violence 18-months later found differences between genders. They found that among boys, cannabis use was associated with a lower frequency of violence (OR 0.6), whereas for girls, the rate was higher (OR 2).

Another study also used structural equation modelling found evidence of a small bidirectional relationship between substance use and delinquency (not specifically aggression), but only in boys and not girls. Notably, the relationship between substance use and later delinquency was only present in the early waves of the study (Mason and Windle, 2002).

A longitudinal study of 702 African Americans followed from age 6 to 42 investigated the relationship between "heavy" adolescent cannabis use (20 or more times in life) and later criminal activity which was measured both by self report, and by examining official criminal records (Green *et al.*, 2010). The authors found that among heavy cannabis users, almost 60% had an arrest record, compared to 35% were non non-users or light users, which included 35% of heavy user who had an arrest for a violent crime, compared with 17% of non/light users. However, after adjusting for possible confounders there was no significant relationship between heavy cannabis use and violent crime. It was however associated with other negative outcomes, such as dropping out of school, spending time in prison, being arrested for a drug offence, and developing a drug diagnosed drug disorder.

Latent class analysis of trajectories of violence from the Project Northland Chicago study (Maldonado-Molina *et al.*, 2010) which followed 3038 12 year old annually for 3 years found 4 classes, "*non aggressive*" "*escalators*", "*chronic aggressive*" and "*desistiors*" (see Chapter 3). Cannabis use was not associated specifically with any of the groups.

The study known as the Woodlawn Study recruited African-American children age 6-7 from a social deprived area of Chicago, and interviewed them again age 16, 32, and 42. Data was analysed from 702 youths who were interviewed at age 16 and again on at least one occasion. The association between heavy cannabis use before age 16 (defined as having used cannabis on 20 or more occasions) and any subsequent violent crimes up to the age of 42 was investigated. Cannabis use was not measured other than at age 16. The authors found no relationship between heavy cannabis use and violent offences, but cannabis use was associated with property and drug offences Woodlawn Study (Green *et al.*, 2010).

It can be seen therefore that there is weak evidence of an association between cannabis use and violence, however little evidence to suggest a causal relationship. Many of the studies that have investigated the association have been beset with similar problems as those that have investigated the relationship between alcohol use and violence, and therefore there is a need for adequately powered longitudinal studies within the general population in which it is possible to control for a comprehensive set of potential confounding factors.

SUMMARY OF FINDINGS AND UNANSWERED QUESTIONS

In summary, laboratory studies in animals and in humans show that the administration of nicotine reduces aggressive responses, and nicotine withdrawal among smokers increases irritability, particularly among those who are have higher trait irritability. Despite the frequency of use in the general population, there are virtually no longitudinal studies that have investigated the relationship between cigarette smoking and violence. The only identified study showed no relationship between smoking and violence. However, this study was limited as it only measured cigarette smoking at baseline to predict violence 18 months later.

Only six studies were identified that specifically reported the longitudinal relationship between cannabis use and violence. The findings were mixed; two study found some evidence of a positive relationship (Mulvey *et al.*, 2006, White *et al.*, 1999), three found no relationship (Green *et al.*, 2010, Pedersen and Skardhamar, 2010, Wei *et al.*, 2004), and one study found that males cannabis users were less likely than male non-cannabis users, and female cannabis users more likely than female non-cannabis users to be violent(Blitstein *et al.*, 2005). The same

criticisms apply as outlined in Chapter 3, that many of the studies are from highly selective samples, and have differing definitions of violence, different times of measurement of outcome and exposure, have only measured the substance (cigarette smoking or cannabis use) at baseline, and have not adequately controlled for confounders.

The following fundamental questions remain unanswered from the data available:

Is cigarette smoking associated with violence in the general population?

Is cannabis use associated with violence in the general population?

Table 3 Summary of Longitudinal Studies that have Investigated the Relationship Between Cannabis and Violence

Name and description	Description	Violence measure	Cannabis Measure	Variables adjusted for	Findings	Comments
The National Youth Survey (Elliott, 1994) Country: USA	1725 11-17 year olds interviewed over 8 annual waves.	Serious violent offences (aggravated assault, robbery, rape that involved injury or use of weapon.	Not reported.	None.	Among those who were violent and reported using cannabis, the initiation of cannabis consumption preceded the initiation of serious violence in 63%.	1. Study designed to investigate the temporal sequence of initiation of alcohol and violence among those who engage in both. Unable to address questions of causality.
Pittsburg Youth Study (PYS) (White *et al*., 1999) Country: USA	1,517 boys age 7, 10 or 13 followed every 6 months for 3 years then every year for 9 years. analysed 506 boys in 6 waves of annual data from age 13-18.	Any of 6 violent behaviours (carried a hidden weapon, strong arming, attacking with weapon with intent to seriously hurt or kill, hurt / threatening to hurt / forced or attempting to force someone to have sex.	Frequency cannabis use in past year.	Property crime Sexual intercourse frequency Academic achievement Depressed mood ADHD symptoms Poor communication with parents	Cannabis use at age 13 associated with violence age 14-18 (OR 5.4), however in 4 out of 5 analyses investigating cannabis use at one year and violence the next showed no significant association.	1. Nationally representative prospective study 2. Analysis not using specific longitudinal methods (series of binary comparisons from one year to the next using dichotomized variables.
TEENS study, Minneapolis (Blitstein *et al*., 2005) Country: USA	2355 students in Minneapolis age 13 followed up 18-months later.	How many times in last year engaged in one of 5 violent behaviours (carry a weapon, hit or beat up someone, group fighting, hurt someone badly enough to need bandages or a doctor, use knife or gun to get something from someone).	How many times in the last 30 days have used cannabis.	Ethnicity Gender Depressive symptoms Influence of spirituality Future onlook Parenting style Cigarette smoking Illicit drug use.	Male cannabis users 0.4-0.9 times as likely to be violent/ Female cannabis users 1.2-3.5 times as likely to be violent.	1. Not a general population study (sampled from low income population). 3. Main purpose of analysis was to determine if predictors of violence differed between boys and girls. 4. Cannabis use other than at baseline was not measured.
Pittsburg Youth Study (PYS) (Wei *et al*., 2004) Country: USA	1517 boys age 7, 10 or 13 followed every 6 months for 3 years then every year for 9 years. Analysed 503 boys age 7 over 6 waves until age	Frequency of cannabis use.	Any of 6 violent behaviours (carried a hidden weapon, strong arming, attacked with weapon with intent	Property crime Sexual intercourse frequency Academic achievement Depressed mood	Violence associated with cannabis use in 5 of the 7 paired analyses in unadjusted anlalyses (OR 2.7-3.8).	1. Relatively small sample (503) 2. Included only boys 3. Series of paired analyses rather than specific longitudinal methods.

	20		to seriously hurt or kill, hurt / threatened to hurt / forced or attempted to force someone to have sex.	ADHD symptoms Poor communication with parents Hyperactivity / inattention / impulsivity Poor communication with parent Poor supervision Parent perception of bad neighbourhood Ethnicity	Aggregated date showed no evidence of relationship once adjusted for potential confounders.	
Woodlawn Study (Green *et al*., 2010) Country: USA	Prospective study of all 1,242 first grade pupils (age 6-7) in Woodlawn community in Chicago in 1966. Interviewed again at age 16, 32 and 42. 702 participants included in analysis.	Heavy cannabis (20 or more times in life) assessed at age 16.	Self reported violent crime. Official convictions of violent crime. Self-reported serious violent behavior	Mothers' School achievement Socio-economic status Family discipline Family activities Family structure Family mobility Family drug and alcohol use Childhood personality characteristics Adolescent substance use Adolescent delinquency School dropout	No association between heavy cannabis use prior to age 16 and official violent crime (OR 0.7-2.1) or self-reported violent crime (OR 0.9-2.1) up to age 42. Higher rates of subsequent drug and property crimes found in cannabis users.	1. Cannabis use after age 16 was not investigated.
Young in Norway Longitudinal Study (Pedersen and Skardhamar, 2010) Country: Norway	1,353 13 year olds in population-based sample followed at age 15, 20 and 27.	Self reported criminal offences.	Self reported frequency of cannabis use during preceding 12 months. Number of times intoxicated with alcohol, number of alcohol-related problems.	Socio-economic status Parental support / supervision Family structure Education level Previous criminal charges Conduct problems History of co-habitation / marriage Alcohol use Other drug use	No association between cannabis use at age 15 and non-drug related offences at age 15-20, or cannabis use at age 20 and non-drug related offences age 20-27 after controlling for confounders.	1. Violent offences not specifically reported.
High risk community psychiatric patients	Prospective study of 517 attendees of emergency	Whether used cannabis or not	Number of incidents of serious violence	None reported	Violence more likely to occur on days preceded	1. Not a representative sample.

in Northeastern USA (Mulvey *et al*., 2006) Country: USA	room of psychiatric hospital in USA who had a recent history of heavy alcohol or drug use and violence. Followed up of 26 weekly interviews		(physical injury, threat with weapon, use of weapon, sexual assault)		by cannabis use (OR 1.2-2.0).	2. No adjustment for confounding.
TEENS study, Minneapolis (Blitstein *et al*., 2005) Country: USA	2355 students in Minneapolis age 13 followed up 18-months later.	How many times in last year engaged in one of 5 violent behaviours (carry a weapon, hit or beat up someone, group fighting, hurt someone badly enough to need bandages or a doctor, use knife or gun to get something from someone).	How many times in the past 24 hours, and in the past 7 days have smoked tobacco. Measure was dichotomized into non/low-level smokers versus weekly or greater smokers.	Ethnicity Gender Depressive symptoms Influence of spirituality Future onlook Parenting style Cigarette smoking Illicit drug use	No relationship between smoking and violence.	1. Not a general population study (sampled from low income population). 2. Main purpose of analysis was to determine if predictors of violence differed between boys and girls. 3. Smoking other than at baseline was not measured.

CHAPTER 5 POTENTIALLY CONFOUNDING VARIABLES IN THE RELATIONSHIP BETWEEN ALCOHOL MISUSE AND VIOLENCE

As already indicated, one model to explain the observed association between alcohol and violence is that each arises from predisposing factors which are common to both, so the relationship may be confounded by these variables, whether inherent to the individual or to his/her social context. It is possible that differences in findings in previous studies can be explained partly by the failure to adequately adjust for confounding. Here I summarise evidence for the potential confounding effects of those variables most consistently associated with violence and/or alcohol in twenty longitudinal studies of offending: age, ethnicity, sex, intelligence, delinquency, personality and temperament, other substance use, peer substance use, family factors, and neighbourhood factors.

AGE

Violence

Crime is very common in the general population; around 40% of individual have a conviction by age 40, but the majority of crimes are committed by only a small proportion of criminals (Farrington, 2014). Studies indicate that delinquency peaks in adolescence and then declines thereafter (Moffitt, 1993), except in around 5% of individuals who continue their delinquency into adulthood and become chronic offenders. Analysis of data from the National Youth Survey showed that, two thirds of participants offended at a low rate, but showed a peak of aggression at age 16 then declined; a smaller group of nearly 12% had an adolescent onset of violence which peaked at age 18 then declined, and a group of a similar size showed onset of violence in young adulthood (age 21) having had no previously reported violence (Nash and Kim, 2006).

Overall it appears that the greatest risk of violence is during adolescence within the population, and except in a minority, the rates reduce with age.

Alcohol

The rates of alcohol consumption increase steadily through adolescence and increases sharply in early adulthood. One of the largest surveys to date was carried out in 1992 in which the US Census Bureau surveyed nearly 43,000 randomly selected individuals and enquired as to drinking patterns and age of onset of drinking (Hingson *et al.*, 2001). The survey found that the mean age of commencing drinking alcohol was 19, while 3% had commenced drinking before the age of 14. Patterns of heavy drinking (4 or more drinks in any day within a month) showed a very sharp increase in 20-25 year olds (up to 50% of males report doing so), followed by an almost linear decline, down to round 15% in the over 65s (Jackson *et al.*, 1998).

Several studies show that the earlier the onset of drinking, the greater the likelihood of subsequent heavy drinking, and higher frequency of use (Harolyn *et al.*, 1998, Maggs and Schulenberg, 2005, Saltz and Elandt, 1986, Zeigler *et al.*, 2005).

ETHNICITY

Violence

There is wide variation in rates of violence between countries, for example, the murder rate in the UK and several other European countries is around 1 per 100,000 per year compared with over 50 per 100,000 per year in some South American Countries (United Nations Office on Drugs and Crime, 2014). There is also variation within countries in rates of violence between people of different ethnic origins. Many studies, such as those from the USA (Piquero and Brame, 2008) and UK (Ministry of Justice, 2011) show proportionally higher rates of violence among Black people compared to Whites or Asians, though differences may reflect variation in social, cultural and economic factors rather than ethnicity per se.

Alcohol

Variations in alcohol use, and attitudes to alcohol vary across ethnicity and culture. A comprehensive review of ethnicity and alcohol use in the UK revealed that the topic is complex, and most studies, as with studies of ethnicity and violence, have

not controlled for other factors such as socio-economic status. Nevertheless, there is evidence to suggest variation in drinking patterns by ethic group; the age of first alcohol consumption is higher among Asian than White or Black ethnic groups, and the rate of increase in consumption during adolescence is greater for White than other ethnic groups (Hurcombe *et al.*, 2010). The frequency of use is also greater among White than other ethnic groups, with Asians generally having much lower alcohol use. Studies in the USA have also found higher rates of alcohol use among White than other ethnic groups (Dawson *et al.*, 1995).

SEX

Violence

Overwhelmingly, studies show that males commit more violent offences than females. For example, 90% of all homicides in the USA are committed by males, and The National Crime Survey in the UK reported that 80% of violent offences are committed by males (Office for National Statistics, 2012b).

Alcohol

Studies consistently show that men drink more alcohol than women. A large survey in the UK reported that 66% of men and 54% of women over the age of 16 had consumed alcohol in the previous week (Office for National Statistics, 2011). Men were also drank more often than women; 16% of men and 9% of women reported drinking on 5 or more occasions in the last week. 39% of men compared with 27% of women drank above the recommended levels on at least one occasion in the past week. There is evidence however that the prevalence of drinking alcohol is equal between boys and girls under the age of 16 is roughly equal. The proportion who reported drinking alcohol increased with age from approximately 11% of 12 year olds to 74% of 15 year olds of both sexes (Health and Social Care Information Centre, 2014a).

INTELLIGENCE

Violence

Intelligence has been extensively studied in relation to offending, and has consistently been found that at a group level, offenders have lower intelligence than non-offenders. Furthermore, numerous longitudinal studies have found that low intelligence and attainment measured in early childhood predicts both juvenile and adult violent offending, e.g. (Denno, 1990, Farrington, 1989, Schweinhart *et al.*, 1990) independent of other risk factors such as socioeconomic status and family history.

Alcohol

There is a highly replicated finding that, unlike the relationship between IQ and violence, there is a positive correlation between IQ and alcohol consumption (Belason and Hafer, 2013, Muller *et al.*, 2013). Longitudinal studies also show that more intelligent children grow up to consume alcohol more frequently and in higher quantities in adult life, even after adjusting for income (Kanazawa and Hellburg, 2010).

DELINQUENCY

Violence

The ordinary dictionary definition of delinquency is "minor crime, especially that committed by young people". Measures of delinquency used in studies include reports of criminal offences, but many studies, depending on the age of the participants, also include behaviours such as truancy and running away from home. Self reported delinquency, involvement in other antisocial behaviours such as stealing and vandalism and drug selling are associated with a greater risk of violence (Farrington, 1989). In a systematic review and meta-analysis of risk factors for youth violence, involvement in low-level delinquency was a very strong risk factor for subsequent violence (Lipsey and Derzon, 1998).

Although studies show an association between violent and non violent offending, there is a debate as to whether violent offending is simply part of a general

tendency for criminal behaviour (Laub and Sampson, 2003) or whether they are different (see Chapter 3). There is in fact evidence to suggest that there may be different and distinct pathways of violent and non-violent offending (LeBlanc and Loeber, 1998, Tremblay *et al.*, 2004).

Alcohol

Drinking alcohol (not under parental supervision) in adolescence is strongly associated with delinquency, and indeed is included in measures of conduct problems or delinquency, such as the Child Behaviour Checklist (CBCL) (Achenbach, 1991). Numerous studies have shown a relationship between early alcohol consumption and delinquency.

PERSONALITY AND TEMPERAMENT

Violence

Many aspects of personality and temperament have been linked to violent behaviour. Much of the early work on personality and crime was carried out using Eysenck's tri-dimensional theory of personality, which defines personality in terms of Extraversion (E), Neuroticism (N) and psychoticism (P) personality traits. A review of studies that investigated personality relating to Eysenck's model of personality and offending showed that high E was related to self-reported offending, and high N was related to official reports of offending. Investigation of the individual questions found that it was the questions relating to impulsivity that largely explained the relationships, leading to the conclusion that research on the link between Eysenck's personality dimensions and offending mainly identified the link between impulsiveness and offending (Farrington and Walsh, 2007). Impulsive acts have been described as poorly conceived, prematurely expressed, unduly risky or inappropriate to the situation that often results in undesirable consequences (Daruna and Barnes, 1993). Impulsivity is itself a multifactorial construct, characterised by at least two core facets, the inability to exert inhibitory control of impulses, and "*delay discounting*", the preference for immediate gratification at the expense of longer-term gain. Impulsiveness was found in the Pittsburgh Youth Study to be strongly associated with delinquency (White *et al.*, 1994).

Many studies have found relationships between the temperaments of young Children and later offending. Children rated as being uninhibited (as opposed to inhibited) at 21 months were significantly more likely to be rated as aggressive at age 12 by both self and teacher reports (Schwartz *et al.*, 1996). In the Dunedin longitudinal study, children rated as being "*undercontrolled*" (restless, impulsive and with poor attention) were significantly more likely to be aggressive, have convictions, and engage in delinquent behaviours at age 18-21 (Caspi, 2000).

Dissocial personality disorder (WHO, 2011) and antisocial personality disorder (American Psychiatric Association, 2000) are associated with violence and criminal behaviour. This is unsurprising as aggression is one of the diagnostic criteria in both.

The five-factor model of personality (FFMP) has become establishes as the most prominent and highly replicated model of personality structure (e.g. (Costa and McCrae, 1992, Donnellan *et al.*, 2006, Funder, 2001, Jones *et al.*, 2011, O'Connor, 2002)). It was originally derived from linguistic trait research, and stemmed from the assumption that individual character differences that are most salient and socially relevant in people's lives will eventually be encoded into their language; the more important such a difference, the more likely it is to become expressed as a single word (John *et al.*, 1988). The initial attempts at deriving categories of personality domains began from creating an exhaustive list of English words to describe personality characteristics (Allport and Odbert, 1936). The list was then condensed and individuals were rated on these personality descriptors. The ratings where then factor analysed (Cattell, 1943, 1945). A total of twelve factors were originally derived, however subsequent reanalyses and replications revealed five factors; these five factors have proved to be very robust and highly replicated personality domains.

While there is general agreement on the number of necessary factors (five), the exact meaning of the factors has been subject of much debate. (Digman, 1990). Factor I is generally accepted to be "*Extraversion/Introversion*" and represents gregariousness, activity, social adaptivity and assertiveness. Factor II is accepted to be "*Agreeableness*", and represents altruism, nurturing, conformity, and likeability and caring at one end of the spectrum, and hostility, self-centeredness, spitefulness and jealousy at the other end. Factor III is "*Conscientiousness*" and is described as incorporating dependability, will to achieve, and responsibility and self-control at

one end of the spectrum, and impulsivity, poor self-control and lack of will to achieve on the other. Factor IV is usually referred to as "*Neuroticism*", and represents a dimension of tendency to experience changes is affect and irritability on one end of the spectrum and emotional stability on the other. Factor V represents "*Openness to Experience*" and incorporates a spectrum of low to high intellectual curiosity, awareness of inner feelings, openness to new ideas and intellectual flexibility.

Research suggests that several personality traits are associated with aggression. Trait aggression (also measured by questionnaire) is correlated with low agreeableness (Miller *et al.*, 2009, Tremblay and Ewart, 2005) and with Emotional Stability/ Neuroticism (Sharp and Desai, 2001, Tremblay and Ewart, 2005). It appears however that neuroticism has stronger correlations with anger (r=0.51) and hostility (r=0.61) than with physical aggression (r=0.26) and verbal aggression (r=0.20) (Sharp and Desai, 2001). Several studies have found a positive relationship between neuroticism/emotional instability and aggression (Buss and Perry, 1992, Caprara *et al.*, 1985, Harkness *et al.*, 1995, Lynn *et al.*, 1989).

An inverse association between agreeableness and aggression/violence is the most strongly and consistently reported personality trait associated with violence (Barlett and Anderson, 2012, Gleason *et al.*, 2004, Heaven, 1996, Miller *et al.*, 2009, Tremblay and Ewart, 2005). Neuroticism is also positively associated with aggressive behaviour (Sharp and Desai, 2001, Tremblay and Ewart, 2005) and aggressive emotions (Barlett and Anderson, 2012). The mechanism of the relationship between agreeableness and violence is thought to be different to the mechanism of the relationship between Neuroticism and violence. Low agreeableness is thought to operate through instrumental or callous hostility, whereas neuroticism is thought to operate through defensive and emotional reactions(Egan, 2009).

Anger is also associated with aggression, as demonstrated in both clinical (Reagu *et al.*, 2013) and non-clinical studies(Berkowitz, 2001, Scarpa and Raine, 1997). Trait anger is defined as the dispositional tendency to experience a wide range of situations as annoying or frustrating, and to have a tendency to respond frequently with elevations in intensity of anger. Anger is not one of the five primary personality traits, but is strongly correlated with neuroticism within the five factor model (Sharp and Desai, 2001, Tremblay and Ewart, 2005), and indeed at least one

model of the factor structure of personality places anger as a sub factor, or facet, of neuroticism (Costa and McCrae, 1995).

Alcohol

Individuals who are diagnosed with an alcohol misuse disorder are consistently found to have high impulsivity as measured in a variety of ways, including laboratory paradigms designed to measure response inhibition (Kollins, 2003, Petry, 2002), and by self-report. For example, a prospective study of over 5000 Finnish hospital staff showed that self-reported impulsivity was associated with both alcohol and cigarette smoking, and higher impulsivity was associated with initiating smoking and becoming a heavy drinker. Although some laboratory studies have shown an association between alcohol intoxication and aggressive behaviour (Bushman and Cooper, 1990), however few studies have investigated the complex interactions between alcohol intoxication and the multifaceted construct of aggression (Oscar-Berman and Marinković, 2007). This is partly because of variation in the definition of aggression in studies, which may include combinations of factors such as impaired decision making, impulsivity, impairment in executive functioning and disinhibition.

A meta-analysis of 20 studies, with a combined sample size of 7,886 participants showed that overall, alcohol use was associated with low conscientiousness (r=-0.22, 95% CI -0.28, -0.17), low agreeableness (r=-0.17, 95% CI -0.21, -0.13) and high neuroticism (r=0.15, 95% CI 0.08-0.22)(Malouff *et al.*, 2007). Effect sizes however were small, and explained only 5%, 3% and 2% of the variance in alcohol use respectively. An interesting sensitivity analysis of the data showed that low agreeableness was associated with alcohol use only in cross-sectional studies, and not longitudinal studies; low agreeableness did not predict later alcohol use, but correlated with current use, suggesting that that alcohol use leads to low agreeableness scores. The data also showed that there was a stronger relationship between neuroticism and alcohol use, in clinical samples (those treated for alcohol use disorders) than in general population samples in which the dependent variable tended to be "ever used alcohol" among juveniles.

The association between personality characteristics and alcohol is different in the case of alcohol use compared with alcohol misuse or dependence. Overall, it appears that the only consistent association with both alcohol use and misuse is an inverse relationship with conscientiousness (Ibanez *et al.*, 2010, Kashdan *et al.*,

2005, Malouff *et al.*, 2007, Martin and Sher, 1994, Trull and Sher, 1994). In the case of alcohol misuse or alcohol disorders, neuroticism is consistently associated (Cooper *et al.*, 2000, Lackner *et al.*, 2013, Ruiz *et al.*, 2003, Stewart *et al.*, 2001, Trull and Sher, 1994). Neuroticism is not associated with alcohol use or misuse in non-clinical samples (Cooper *et al.*, 2000, Kashdan *et al.*, 2005, Paunonen and Ashton, 2001, Peterson and Morey, 2005).

Extraversion has been shown to be associated with alcohol use in non-clinical populations, in alcohol use disorders (Cooper *et al.*, 2000, Malouff *et al.*, 2007, Peterson and Morey, 2005) and alcohol problems (Ruiz *et al.*, 2003, Stewart *et al.*, 2001, Vollrath and Torgersen, 2002), but not in clinical populations ((Malouff *et al.*, 2007, Martin and Sher, 1994, Trull and Sher, 1994). Openness and agreeableness have been fount to be inconsistently associated with alcohol use or alcohol use disorders (Ibanez *et al.*, 2010, Lackner *et al.*, 2013, Malouff *et al.*, 2007, Ruiz *et al.*, 2003).

One of the studies included in the meta-analysis also investigated family history of alcoholism (Martin and Sher, 1994). Family history of alcoholism was positively associated with openness and negatively associated with agreeableness and conscientiousness.

With regard to smoking, a similar pattern of associations with personality characteristics was found in a meta-analysis of 9 studies involving 4,730 participants, which investigated associations between personality traits and smoking. The meta-analysis showed that smoking was associated with low conscientiousness (r=-0.22), low agreeableness (r=-0.20) and neuroticism (r=0.23)(Malouff *et al.*, 2006).

Rather going against the conceptualization of both personality traits and substance use being both on a continuous spectrum, Chassin and collegues (Chassin *et al.*, 2004) chose to impose a categorical structure onto longitudinal trajectories of alcohol misuse using growth mixture modelling. Their resulting analyses described three latent classes of alcohol misuse, which corresponded to “abstainer”, “medium” and “high” alcohol use. They found that openness was positively associated and conscientiousness was negatively associated with the heavy drinking group, and agreeableness was associated with the abstainer group.

Personality characteristics also appear to predict response to treatment and relapse among those with alcohol dependence. Two studies have found that relapse rates were significantly higher among those with baseline high neuroticism and low conscientiousness (Bottlender and Soyka, 2005, Fisher *et al.*, 1998).

Conscientiousness and agreeableness were also been found to be negatively correlated with total weekly alcohol consumption consumed amongst a sample of 142 University students (Clark *et al.*, 2012).

In summary, there is evidence that specific personality traits are associated with both alcohol use and aggression, especially agreeableness and neuroticism. However, it is not clear how or to what extent those personality characteristics may underpin the relationships between alcohol misuse and violence.

OTHER SUBSTANCE USE

Violence

There is evidence that illicit drug use is associated with violence. In England and Wales 12% of people arrested for assault tested positive for cocaine or illicit opiates (Bennett, 2000). Numerous studies have linked illicit substance use to rates of interpersonal violence (Stuart *et al.*, 2009), and several studies have found that specific drugs, particularly benzodiazepines, cocaine, crack cocaine and stimulants can increase aggression in experimental settings (Ben-Porath and Taylor, 2002, Kuhns, 2005, Roth, 1994).

Alcohol

It has been well established that young people who drink alcohol, especially those who drink more heavily, are also more likely to smoke and use other drugs (Kanazawa and Hellburg, 2010, Yu and Williford, 1992). According to the National Survey of Drug Abuse (NIDA) in the USA in 2009, adolescents age 12-17 who were categorised as heavy alcohol consumers were thirteen times more likely to use illicit substances than non-drinkers (Abuse, 2012).

PEER SUBSTANCE USE

Violence

Involvement with peers who are delinquent is associated with violence. Data from the Christchurch birth cohort showed that affiliation with delinquent peers was associated with violent crime (Fergusson *et al.*, 2002). One study from the Chicago Youth Development Study however showed that violence was associated with having violent, but not non-violent delinquent peers (Henry *et al.*, 2001). In a systematic review and meta-analysis of risk factors for youth violence, peer antisocial behaviour had the second largest effect size (0.37) among 12-14 year olds (Lipsey and Derzon, 1998).

Alcohol

Studies have shown that peers attitudes to alcohol and drinking behaviour has a very significant influence on the attitudes and drinking patterns of children, even more than that of their parents (Ary *et al.*, 1993, Newcomb and McGee, 1989). Affiliation with deviant peers has also been shown to mediate the relationship between harmful drinking and depression in adolescents (Pesola *et al.*, 2015). However, Several studies however have shown that alcohol can also have a positive impact on peer relationships in terms of peer bonding and social integration e.g. (Brown *et al.*, 2008).

FAMILY FACTORS

Violence

A comprehensive review of factors associated with offending in males found that poor parental child management techniques and parental offending were two of the factors that were most strongly associated with delinquency (Loeber and Dishion, 1983). A review of family factors associated with offending found that the factors with the strongest relationships (in order) were poor parental supervision, parental rejection of child, large family size, low parental involvement with children, parental conflict and antisocial parents (Hoeve *et al.*, 2009, Loeber and Stouthamer-Loeber, 1986).

Alcohol

The effects of the family on childhood drinking and subsequent alcohol consumption of the offspring in adulthood has been widely studies but has produced mixed findings. Many studies have reported that parental alcoholism increases the risk of alcohol problems in the children (Brennan *et al.*, 1986a, b, Patton, 1995) and that parental attitudes to alcohol and their alcohol use is modelled by their children (Ary *et al.*, 1993). However this finding is not unanimous, as some studies have shown no relationship once social and economic factors are taken into account (Berkowitz and Perkins, 1986). The mechanism of the relationship is therefore likely to be multifactorial. Some studies have concluded that the relationship between the parent and child is affected by the parent's drinking, which disrupts that development of emotionally stable children, and in turn influences the child's drinking. Disrupted families, and poor or conflictual relationships within the family have been shown to increase the likelihood of early initiation and alcohol problems in the children (Berkowitz and Perkins, 1986, Hawkins *et al.*, 1992), while the opposite has been shown to be protective (Denton and Kampfe, 1994).

NEIGHBOURHOOD FACTORS

Violence

Many studies have investigated the variation of rates of offending between communities. Studies have found that communities with high levels of poverty and disadvantage have higher rates of crime (Loeber *et al.*, 2005, Sampson *et al.*, 1997). Higher population density has also been shown to be associated with crime (Huizinga *et al.*, 2003, Osborn, 1980).

Alcohol

There is conflicting evidence as to the role of neighbourhood levels of disadvantage on rates of alcoholism. For example, one study has shown that teenage boys but not girls who live in neighbourhoods with high levels of unemployment have higher levels of alcohol consumption (Karvonen and Rimpela, 1997), and a multilevel analysis of adults showed that the most deprived areas were associated with the highest levels of alcohol consumption (Pollack *et al.*, 2005, Rice *et al.*,

1998) whereas other studies have failed to find an effect of the neighbourhood once individual characteristics are taken into account (Ecob and Macintyre, 2000, Rice *et al.*, 1998).

There is a relationship between alcohol availability (measured as the density of alcohol outlets) and violent crime (Gorman *et al.*, 2001, Livingston, 2008a, b). There is also evidence that the level of neighbourhood disorganisation (including the perceived amount of crime in the neighbourhood) is associated with alcohol and drug problems among adolescents (Winstanley *et al.*, 2008) although it is unclear at to the direction of the relationship.

It has been suggested that there is variation in patterns of drug and alcohol use between rural and urban areas, and that the rural environment may be a protective factor with regard to drug and alcohol exposure (Eberhardt *et al.*, 2001). Few studies have investigated this, however a large survey of over 14,000 high school students in the USA found, with the exception of drunk driving which was higher in rural populations, no differences in the prevalence of alcohol related risk behaviours between rural, urban, suburban and rural settings (Greggo *et al.*, 2005).

CHAPTER 6 AIMS OF THESIS

The main purpose of this thesis is to examine in detail the relationship between the use of alcohol and the risk of serious violence in young people in the general population. I have used data from a nationally representative cohort study to investigate whether consumption of alcohol is an independent risk factor for serious violence after adjusting for a comprehensive set of confounders. The second purpose of this thesis is to investigate the relationship between cigarette smoking and cannabis use and violence in the general population.

Although there is evidence that alcohol and violence are associated, and that those who drink alcohol at a young age are more likely to engage in other problem behaviours, the evidence from previous research as to whether and to what extent alcohol is an independent risk factor for violence is equivocal; differences in findings are likely to be due to variations in the definitions of violence, differences in age of participants, highly selective samples, variations in length of time between exposure and measurement of outcome, and failure to account for changes in both exposure and outcome over time.

The first aim of the thesis therefore is to investigate whether alcohol use is associated with serious violence independent of the effects of other risk factors, including delinquency, IQ score, personality characteristics, family structure and neighbourhood characteristics that could confound the relationship. I wished to investigate the relationship in a large nationally representative general population sample, and to eliminate as far as possible reverse causality by using a study with a prospective longitudinal design.

High amounts of alcohol consumption including binge drinking appear to be associated more strongly with violence than lower levels of alcohol consumption. It is not clear whether drinking frequency, volume or both are equally important in the possible relationship with violence. My second aim was to investigate whether different patterns of alcohol consumption are associated with serious violence. I hypothesized that quantity of alcohol would more likely be associated with violence than frequency of consumption.

It is possible that younger people are more susceptible to the effects of alcohol than older ones, and my third aim is to test the extent to which the effect of alcohol on violence varied with age.

Cigarette smoking is frequently associated with delinquency and other problem behaviours among children and adolescents. Laboratory studies indicated that nicotine reduces aggression, but nicotine withdrawal increases irritability. Despite the high prevalence of smoking in the general population the extent to which smoking is independent associated with violent in either increasing or reducing the risk is unknown. My fourth aim of this thesis is to investigate whether cigarette smoking is associated with violence independent of other risk factors, and my fifth aim is to investigate whether there was variation according to age.

My sixth aim is to investigate whether cannabis use was associated with violence. Conflicting evidence exists from previous studies that have investigated cannabis use and violence. I aimed to investigate the extent to which, after adjusting for a comprehensive set of confounders that cannabis use was independently associated with violence among adolescents and young adults. As adolescent brains are still maturing and undergoing an intensive period of development they may be more susceptible to the effects of cannabis. Therefore, as with the investigations of alcohol and cigarette smoking, my seventh aim is to investigate the extent that the effect of cannabis use on violence may vary with age, independent of other risk factors, and taking into account changes in cannabis and other substance use.

Some studies have indicated that the effect of alcohol on violence varies according to underlying personality characteristics, for example that those who consume alcohol and are violent are those who are dispositionally aggressive. The most widely supported model of personality contains five factors, extraversion, agreeableness, conscientiousness, neuroticism and openness. There is evidence that specific personality traits are associated with both alcohol use and aggression, especially agreeableness and neuroticism. However, it is not known how or to what extent that personality characteristic may underpin the relationships between alcohol misuse and violence. My final aim is to investigate whether alcohol mediates the relationship between specific personality traits and violence, and undertake an evaluation as to which individual, family and neighbourhood factors confound the relationship between alcohol and violence.

SUMMARY OF AIMS

To investigate the relationship between alcohol use and the risk of violence during adolescence and early adulthood in the general population.

To investigate the relationship between frequency or quantity of alcohol use and risk of violence.

To investigate whether the relationship between alcohol use and violence varies with age.

To investigate the relationship between cigarette smoking and risk of violence during adolescence and early adulthood.

To investigate whether the relationship between cigarette smoking and violence varies with age.

To investigate the relationship between cannabis use and risk of violence.

To investigate whether the relationship between cannabis use and violence varied with age.

To investigate whether alcohol may mediate the relationship between each of the five personality traits extraversion, agreeableness, conscientiousness, neuroticism and openness and violence.

SECTION II METHOD

CHAPTER 7 EPIDEMIOLOGICAL METHODOLOGY

Before going on to describe the current investigation in more detail, I will first provide an overview of the statistical methods employed, and the reasons for the choice of those methods.

CAUSAL INFERENCE

Although the purpose of epidemiological studies may be to establish evidence of a causal relationship between exposure and outcome, it is the association between exposure and outcome that is measured. Before evidence of association is put forward to support evidence of causation, alternative explanations for the association should be explored. They include:

Chance –the random spurious association between the variables.

Confounding – where an extraneous variable that is correlated with both the exposure and outcome, affects the apparent relationship between the two.

Bias – where systematic error arising from either the design or execution of the study, which produces an incorrect estimate of the association between exposure and outcome.

Reverse causality –when the exposure occurs as a consequence of the outcome (rather than the exposure causing the outcome).

Once the validity of the association has been explored, further consideration as to whether the association can be considered causative should be undertaken. The Bradford-Hill criteria (Bradford-Hill, 1965) for causality provides a framework for such consideration, and has 9 parts:

Temporality – The exposure must come before outcome;

Strength – A strong relationship between exposure and outcome indicates that the relationship is less likely to be due to an extraneous variable;

Biological gradient (or dose-response relationship) such that there is a direct relationship between the magnitude of exposure and magnitude of outcome;

Consistency – the association is consistently found after multiple replications;

Plausibility – where there is a rational or theoretical basis for the relationship;

Coherence – the relationship does not conflict with current knowledge;

Analogy – where an accepted phenomenon in one area can be applied to another area;

Experiment – Association found in experimental rather than observational studies;

Specificity – Demonstrating that the effect has only one cause.

COHORT STUDIES

A cohort study is a longitudinal observational study in which a group of people who share a common characteristic (the cohort) are observed in at least one future time point for the presence of the outcome of interest. The cohort is selected on the basis that none have the disease of interest at baseline, and are classified according to exposure to a suspected risk factor for the disease. A comparison is then made between the incidence of outcome in those exposed versus those not exposed to the risk factor.

There are three different type of cohort study: (1) The classical cohort study in which the groups to be studied are selected because of their exposure and are followed up to compare the incidence of the outcomes of interest; (2) The population cohort study in which a group of individuals is selected due to particular circumstance (for example a birth cohort, or a cohort of school children). Multiple exposures and multiple outcomes can then be studied; (3) the historical cohort study in which the exposures where measured for other purposes before the study was commenced.

Cohort studies allow for the temporal sequence of events to be investigated because the exposure is defined before the outcome has occurred. The design

therefore allows inferences to be made in relation to causality. The design is also less susceptible to the effects of information bias in the ascertainment of the exposure status, because the exposure status is determined before the outcome has occurred. Cohort studies are useful in studying rare exposures, and can be used to study multiple outcomes. Disadvantages of the design are that they may be resource intensive and time-consuming, they are generally unsuitable for the study of rare outcomes, and they are prone to selection bias due to loss to follow-up, and to confounding (Weich and Prince, 2003).

The purpose of the epidemiological study is to estimate findings such as incidence and association in a study sample that can then be generalised to other populations. Study participants should therefore be selected to be representative of the populations to which results are to be generalised.

There are numerous analytical methods for measuring association in cohort studies. Subsequently I will outline the basic method of using odds and odds ratios. I will then go on to describe the statistical methods that are needed for measuring association in longitudinal data where there are repeated measures from individuals, and then go on to describe methods for analysing change.

CONSTRUCTION OF SCALES

Quantities of interest in this study were observed indirectly through quantifying responses to questions posed to participants in the form of standardised questionnaires. In some cases, the variable of interest can be ascertained from the response to a single question, such as the person's date of birth, or the number of siblings they have. These questions relate to values that, in principle, are directly observable, but a questionnaire was used as a convenient method of ascertaining the information. There may therefore be some error in these values due, for example, to a participant's error in reading or understanding the question, or making mistakes when completing the questionnaire. No method of data collection, however, is completely proof against recording errors.

Other quantities of interest cannot be observed directly, such as personality or intelligence. Such quantities relate to an underlying "construct", for which specific measures may be developed. In this study, examples include measures of delinquency and intelligence (see below). Typically, a number of questions, are

asked and then scored answers are combined as a scale to give a continuous measure of the purported construct. The degree to which the scale measures what it purports to be measuring is known as the construct validity (Nunnally and Bernstein, 1994).

Many of the scales within Add Health have been adapted from previously validated scales. An example is the Peabody Picture Vocabulary Test, Revised (Dunn and Dunn, 1981) which has been validated by comparing the results obtained on this scale to those on a previously validated "gold standard" intelligence scale, the Wechsler Adult Intelligence Scale (Craig and Olson, 1991).

When individual questions are combined to form a scale with a single numerical value, this is often done by simply adding the score from each answer. If so, it is necessary that all of the questions have internal consistency - that is that they are measuring the same thing (Bland and Altman, 1997). Therefore, all of the items should be correlated with one another. The internal consistency of items can be measured using Cronbach's alpha(Cronbach, 1951). This is given by:

$$= \frac{k}{k \quad 1}(1 \quad \frac{s_i^2}{s_T^2})$$

where k is the number of items in the scale, s_i^2 is the variance of the *i*th question, and s_T^2 is the variance of the total score formed by summing all if the questions. The principle behind it is that the variance of the sum of a group of independent variables is equal to the sum of their variances. Therefore, if the items in the scale are independent of one another (they are not measuring the same thing), then the variance of the sum of the items, s_T^2, will be equal to the sum of their individual variances, s_i^2, and will be equal to zero. On the other hand, if all of the items are the same, will be equal to 1. For scales that are used for research, an alpha value of above 0.7 is considered satisfactory (Bland and Altman, 1997).

For each of the variables of interest in this study, previously validated scales were used where available. All of the scales were plotted and the distribution inspected, and searched for outliers. The Cronbach's alpha was calculated for each scale as a

check of the internal consistency of the scale. In some cases, scales were divided into ordered categories for further analyses.

Some of the variables, for example temper, impulsivity or social deprivation, I constructed from individual items within the set of questionnaires, and were not based on previously validated scales. For these items, questions were selected first on the basis of face validity; that is they appeared to be asking questions related to the construct of interest. The degree to which the items were correlation was first assessed, and the Cronbach's alpha for the set of questions calculated. In addition, the "item-test" and the "item-rest" correlations were calculated. These refer, respectively, to the degree to which each item correlates with the overall scale, and the degree to which the item correlates with a scale constructed without that item. Items that showed a low correlation with the scale were removed, and the scale was constructed from the remaining items that showed a good correlation, such that the resulting Cronbach's alpha was satisfactory. As a final check, I carried out principal component factor analysis on the scale, to ensure that the resulting scale was unidimensional.

MEASURES OF ASSOCIATION

The probability of an event occurring is the proportion of times an event will occur in a large number of identical repeated trials. This is known as a frequentist definition of probability, as it is derived from a retrospective analysis of the frequency of events.

Probability as it applies to epidemiology is more commonly based on what is know as a subjective definition, in which probability represents the expectation or likelihood of an event occurring. The values of a probability lie between 0 and 1.

Related to probability is the concept of odds. The odds of an event occurring is defined as the probability that an event occurs, divided by the probability that the event does not occur:

$$Odds(A) = \frac{\text{Probability(A occur)}}{\text{Probability (A does not occur)}} = \frac{\text{Prob(A)}}{1 \quad \text{Prob(A)}}$$

The odds are estimated by:

$$\text{Odds} = \frac{p}{1\ \ p} = \frac{d/n}{(1\ \ d/n)} = \frac{d/n}{h/n} = \frac{d}{h}$$

Where d = Number of people who experience specified event

n= Total number in the sample

h= Total number of people who do not experience specified event

p= Probability of specified event occurring

The odds of an event is always larger than the probability, however the values tend towards being equal as the values get smaller. The odds can lie between 0 and ∞.

ODDS RATIO (OR)

The odds ratio is a measure of effect size that is derived from the ratio of the odds of an event occurring in one group compared with the odds of it occurring in another group.

$$\text{OR} = \frac{\text{odds of disease in exposed group}}{\text{odds of disease in unexposed group}} = \frac{d_1/h_1}{d_0/h_0} = \frac{d_1 \text{x}\ h_0}{d_0 \text{x}\ h_1}$$

d_0=outcome present in unexposed group

d_1=outcome present in exposed group

h_0=outcome not present unexposed group

h_1=outcome not present exposed group

The odds ratio is restricted at the lower end to 0 and therefore has a skewed distribution (Bland and Altman, 2000). The log of the odds ratio has more useful properties for statistical modelling applications. Unlike the odds ratio, the log odds ratio can take any value between -∞ and ∞, and has an approximately normal distribution. Log odds ratios are therefore preferred in statistical modelling.

It is possible to calculate the standard error of a log odds ratio, and hence a 95% confidence interval. A confidence interval is a range of values around an estimate that indicates the reliability of the estimate. A wider confidence interval around an estimate therefore represents less reliability of the estimate than a narrower confidence interval. By convention, 95% confidence intervals are presented for estimates, which represents a range in which there is a probability of 0.95 that the true value of the estimate lies within the range. It is defined as 1.96 standard errors on either side of the estimate. The antilog of the intervals can be taken to give the confidence intervals of the odds ratio.

The standard error of the log odds ratio is estimated by the square root of the sum of the reciprocals of frequencies:

$$\mathrm{SE}(\log OR) = \sqrt{\frac{1}{d_0} + \frac{1}{d_1} + \frac{1}{h_0} + \frac{1}{h_0}}$$

The 95% confidence interval of the log(OR) is given by:

Log(OR) ± 1.96(SE log(OR))

LINEAR REGRESSION

Simple linear regression is a method used to find the equation for the line that best fits the relationship between two variables. It describes how much the outcome (y

in the notation used here) changes with a change in the exposure variable (given as x). The equation is given by:

$$y = \quad + \quad x$$

is the intercept, which is the value of y when x is zero.

is the slope, the change in y for every unit change of x.

As stated, linear regression is a method to find the line that best fits the relationship between two variables. The method used is known as least squares. This is a method to find the line that most closely "fits" the data; that is, in which the observed data points are, overall, as close to the line as possible. To put it another way, the method finds the values for and that minimise the vertical distances from the line (in fact the squared vertical distances). This is given by:

$$= \frac{(x \quad \bar{x})(y \quad \bar{y})}{(x \quad \bar{x})^2}$$

where $\bar{x}$ and $\bar{y}$ represent the mean of x, ($\bar{x} = \frac{x}{n}$), and mean of y, ($\bar{y} = \frac{y}{n}$), respectively.

Multiple linear regression is an extension of the principles of simple linear regression. It is a statistical technique used to measure simultaneously the effect of multiple variables on an outcome, while adjusting for the effects of the other variables on the outcome.

The general equation for multiple linear regression with r exposure variables is an extension of that for simple linear regression, and is given by:

$$y = \quad + \quad _1x_1 + \quad _2x_2 + \quad _3x_3 + \ldots + \quad _rx_r + e$$

In this equation, there is also an error term, e, which is the square root of the residual mean square.

There are two main assumptions that underlie linear regression. The first assumption is that y is normally distributed for any value of x. The second assumption is that the variance around the regression line is equal for all values of the predictor variable, x. This equal variance, also known as "homoscedasticity", simply means that there is the same amount of scatter around the regression line throughout the length of the line.

LOGISTIC REGRESSION

Logistic regression is a statistical method that is commonly used for the analysis of binary outcome variables. It provides a method for analysing the association between multiple exposures simultaneously and a single binary outcome. Logistic regression is based on modelling odds ratios. As in linear regression, it provides the statistical methods for modelling the effect of an exposure on an outcome, but in the case of logistic regression, it is used when the outcome is binary. Like multiple linear regression, it also provides the means of analysing the effect of multiple exposure variables simultaneously. Analogous to the equation for multiple linear regression, the general form of the logistic regression model is given by:

$$\text{Log odds of outcome} = + {}_1x_1 + {}_2x_2 + {}_3x_3 + \ldots + {}_rx_r$$

Analogous to linear regression, represents the intercept and is the value of the outcome when the coefficients of the exposure variables (the s) are zero. The difference between logistic regression and linear regression is that it is the log odds of the outcome that is being modelled, rather than the value of the outcome for a given value of the exposure variable or variables. In the case of the binary

exposure variable, x_1, the method compares the log odds of the outcome in the unexposed group (x_1=0), to the odds of the exposed group (x_1=1).

Logistic regression can also be used with exposure variables measured on a continuous scale to estimate the log odds of the outcome per unit change in exposure, (assuming that the change is the same for each unit change of exposure).

An advantage of logistic regression is that it requires few assumptions; specifically, it is not necessary that there is a linear relationship between the dependent and independent variables, or between the independent variables. Furthermore, it does not need to be assumed that there is equal variance within groups (whether measured on an interval or continuous scale). The categories of the dependent variable must, however, be mutually exclusive and exhaustive, such that every case is a member of (at least and only) one of the groups. A sample size of at least 50 cases per independent variable is also recommended (Field, 2009).

Analogous to modelling odds ratios for comparison of two exposure groups:

Log (Odds outcome) = log(Baseline) + Log (Exposure odds ratio)

Where the baseline odds is the exposure group that is used as a comparison by which all other groups are compared. The exposure odds ratio is the effect of the exposure on the odds of the outcome.

NUMBER NEEDED TO HARM

The number needed to harm (NNH) is a measure of how many individuals would need to be exposed to a risk-factor over a specified length of time to cause harm in one individual who would not otherwise have been harmed. It is defined as the inverse of the attributable risk.

The attributable risk (AR) is the difference in rate of an outcome between an exposed and an unexposed population

AR = Incidence in exposed - Incidence in unexposed

An adjusted NNH can be calculated from the odds ratios and the patient expected event rate (PEER). This is the rate of the outcome among the population not exposed. The adjusted NNH is calculated as follows:

$$\text{NNH}=\frac{(1 \quad ((PEER) \quad (1 \quad OR)))}{(1 \quad PEER) \quad PEER \quad (1\text{-OR})}$$

Confidence intervals for the estimate of the NNH can also be calculated to indicate the degree of uncertainty of the estimate (D Altman BMJ 1998;317:1309).

GENERALISED ESTIMATING EQUATIONS (GEE)

In longitudinal panel data there are, by definition, multiple observations from each individual; within individuals these observations are correlated. One of the assumptions of basic regression, however, is that observations are independent from one another. Use of traditional regression is therefore not suitable for such longitudinal data, as it will result in biased estimates of standard errors.

Traditional regression approaches also rely on the assumption that the errors are normally distributed and homoscedastic (the error term (or random disturbance) in the relationship between the independent variables and the dependent variable is the same across all values of the independent variables.; these assumptions are often not met in practice (Ghistetta and Spini, 2004, Liang and Zeager).

The GEE are an extension of ordinary linear regression and allows for the correlation in observations within individuals. The GEE estimates the average response in the population, and is therefore also known as a population-averaged approach.

The GEE method relies on the assumption that (1) the outcome variable is linearly related to the exposure; (2) the number of clusters (individuals in longitudinal data) is at least 10; and (3) the observations in different clusters are independent. GEEs do not imply any distribution assumptions about the data, and can account for unbalanced data (e.g arising from unequal spacing of waves in longitudinal data), and accommodate missing data. This makes this approach particularly

suitable for applications in analysis of longitudinal data. The link function, or distribution of the outcome variable (e.g. binomial distribution for binary data, Poisson for count data), must be specified, as must the correlation structure (the correlation of residuals within individuals or clusters). An advantage of GEE models is that estimation of standard errors of parameters is robust even when the correlation structure is mis-specified. This is particularly the case when there are large numbers of participants and fewer observations per participant.

GEE models do not measure change specifically (see below), but can be thought of as extensions to ordinary regression methods that adjust for the non-independence of observations within individuals.

MEASURING CHANGE

Longitudinal studies allow for the investigation of how individuals change over time. From a statistical perspective, there are two types of questions that form the core of every study of change: (1) How does the outcome change over time? and (2) Can the differences in these changes be predicted? (Singer and Willett, 2003). These questions are important to my study of the relationship between alcohol and violence.

In order to carry out a multilevel model of change, there are four main characteristics that are necessary:

1. *There must be longitudinal data with three or more waves of data;*
2. *There must be an outcome whose values change systematically over time;*
3. *There must be an appropriate metric for time;*
4. *The value of the outcome on any occasion must be equitable over time; that is, the same measure should be used at each time point.*

With regard to the appropriate measure of time, in the case of cohort studies in which data-collection has been carried out over equally spaced intervals, an appropriate metric of time may simply be the consecutive numbering of data collection occasions (time-point 1, time-point 2 etc). In the case of studies where there is an unequal period of time between data collection waves, the above is unsuitable, and a preferred measure of time may be the amount of time elapsed

since the beginning of the study, such as the number of days or weeks since the first episode of data-collection.

Collection of the Add Health the data (which I have used in my study) was unequally spaced over time (see also below), with the second wave occurring around 1 year after the first, followed by Wave III and Wave IV at an average of 7 and 13 years later. In addition, the cohort was designed to include individuals within an age range of between 12 and 18 at inception of the study. Furthermore, there was variation within the follow-up schedules between individuals, in some cases of up to a year. The appropriate metric of time therefore was the individual's age at each data collection point, rather than the dates of data collection/data collection intervals.

When running a random-effects model for longitudinal data in which the data is clustered at the level of individuals, and there are multiple measures of a variable of interest at different time-points, the process is broken down into two stages. Rather than simply performing a regression of the variable of interest by age over the entire sample, the first stage is to fit individual linear regression models for every individual in the sample. This produces a regression line for every individual, each with its individual intercept and slope. It is the individual intercepts and particularly their slopes which are of most interest, and which can be used as the object of further analysis. The purpose of this stage is to describe within-individual change over time. The equation for this stage is known as the "level-1" sub-model. In the case of the analysis of a measure of violence, the violence score can be thought of as being plotted on the y-axis, and the age of the individual when the measure was observed on the x-axis. A linear regression line is then fitted through these data points, and this is the person's individual growth trajectory.

The level-1 submodel is described as follows:

$$Y_{ij} = [\ _{0i} + \ _{1i} TIME] + [\ _{ij}]$$

The model assumes a linear relationships that describes each person's true change over time, with any deviation from linearity assumed to be due to random

measurement error or other unobserved factors ($_{ij}$). $_{0i}$ and $_{1i}$ are known as the individual growth parameters, and characterise the hypothesized true trajectory for the ith subject. They are analogous to the population intercept and slope in linear regression, but relate to the individual. The first individual growth parameter $_{0i}$ is the intercept, the true value of Y when time=0. The second individual growth parameter is $_{1i}$, which is the slope of the individual's growth trajectory; it represents the rate at which the given individual changes over time with respect to the variable of interest. The error term, $_{ij}$ represents the vertical distance between the observed data and the fitted regression line. The level-1 residual variance ^{2}, is the net vertical scatter of the observed data around the individual's linear trajectory.

Fitting these level-1 models on every individual allows for every person to have his or her own trajectory, and hence their own individual growth parameters that describe them (intercepts and slopes). These growth parameters then become the object of analysis in the second stage, the "level-2 submodels".

The second stage, involves fitting the level-2 submodel. Whereas the level-1 submodel is concerned with analysing change within individuals, the purpose of the level-2 submodel is to analyse differences in change between individuals, by analysing the individual growth parameters collectively as obtained from the level-1 submodel. It is of particular utility in investigating the relationship between predictors and these growth parameters (intercepts and slopes from the level-1 model, for example to test the hypothesis that the baseline and rate of change of violence is greater in those who drink alcohol compared with those who do not) (Singer and Willett pg 8). Statistical modelling of both these levels is known as the "multilevel model of change".

The level-2 submodels are in two parts and analyse the individual growth parameters (^{0i} and ^{1i} from the level-1 submodel). They are in the form of standard regression equations, but they treat the level-1 growth parameters as outcomes that may be associated with a predictor (such as level of alcohol consumption).

$$_{0i} = _{00} + _{01}PREDICTOR_i + _{0i}$$

$$_{1i} = _{10} + _{11}PREDICTOR_i + _{1i}$$

In these models $_{00}$ represents the average of the level-1 intercepts in the population; it is the value of the predictor at baseline (time=0). $_{01}$ is the population average change in the level-1 intercepts when there is a 1-unit change in the level-2 predictor. $_{10}$ is the population average of the level-1 slopes, $_{1i}$ for those with a level-2 predictor of 0. $_{11}$ is the population average change in level-1 slope when there is a 1-unit change in the level-2 predictor. $_{0i}$ and $_{1i}$ are the error terms and represent deviation between individual growth parameters and their respective population averages. The first of the level-2 equations is concerned with modelling the individual's intercept. It states that the true baseline (intercept) of the outcome for person *i* is equal to the population average baseline (intercept) plus the product of the value of the predictor and the difference in baseline for a 1-unit increase in the predictor, plus the amount of the outcome that is not explained by the predictor. The second of the level-2 equations is concerned with modelling the individual's slope. It states that the true rate of change (slope) of the outcome for person i is equal to the population average rate of change, plus the product of the value of the predictor and the difference in rate of change per unit increase in the predictor, plus the amount of the slope that is not explained by the predictor.

The level-1 and level-2 model can be represented equivalently as a composite model by combining and rearranging the above formulae.

$$Y_{ij} = [_{00} + _{10}TIME_{ij} + _{01}PREDICTOR_i + _{11}(PREDICTOR_i \quad TIME_{ij})]$$

$$+[_{oi} + _{1i}TIME_{ij} + _{ij}]$$

In the case of modelling a continuous measure of violence over time, in which alcohol consumption is the predictor, and the subject's age is used as the measure

of time, this model therefore states the following: the amount of violence for person *i* at occasion *j* is equal to the average population intercept (average population baseline level of violence when alcohol consumption is zero), plus the individual's level of alcohol consumption multiplied by the population average change in the intercept per unit of alcohol consumption, plus the population average slope multiplied by the product of their age and the level of alcohol consumption, plus individual *i*'s difference in intercept from the population average, and the individual's difference in slope multiplied by a product of his/her age and the level of alcohol consumption, plus the total amount of violence that is unobserved and not predicted by his or her age.

In addition, the random-effects model allows one, for a given outcome, to investigate the specific amount of variation within individuals versus that which is between individuals. This is measured using the intraclass correlation coefficient (ICC), which is the ratio of the between individual variance to the total variance, thus if all of the variation was within individuals, the ICC would be equal to one; if all of the variation was between individuals, i.e. there is no evidence of clustering, then the ICC would be equal to zero.

In contrast to fixed-effects models (see below), random-effects models sometimes referred to as mixed models or multi-level models, allow for the changes both within and between individuals to be analysed.

RANDOM- AND FIXED-EFFECTS MODELS

There are two ways of defining the effects of an exposure on an outcome. The first, are the "fixed effects", which are the variables that are of intrinsic interest, and that have the same values over time (for example the values ethnicity). In contrast, the random effects are not usually of intrinsic value, do not have defined values, but represent a source of variation within the sample. This source of variation may arise from clusters within the sample, such as individuals from the same family, school or neighbourhood. In longitudinal data, in which there are multiple measures of variables at different time-points within individuals, each person's observations can be considered to be a cluster, and therefore the "random effect" can be at the individual level.

There are two different methods of analysing longitudinal data, which differ in whether they model only the fixed-effects known as "fixed-effects models", or whether they also model the random effects, known as "random-effects models. I will describe these two approaches in more detail below.

FIXED-EFFECTS MODELS

Fixed-effects models are tools for the analysis of longitudinal data. They may be seen as extensions of methods used in cross-sectional studies, such as linear and logistic regression, but where there are multiple measures of a variable at different time-points within individuals. They are used when change in one variable in relation to another is the object of interest. The underlying assumption in such models is that other individual variables remain constant or, to use the model terminology, fixed.

Fixed-effects models are therefore designed to study the causes of change within individuals over time. In the case of any relationship between alcohol consumption and violence, which may each change over time, fixed effects models can be used to compare within individuals, rates of violence in relation to changes in alcohol consumption.

In all studies, whether experimental or observational, there is a need to remove, as far as possible, the effect of extraneous variables on the object of interest, so that conclusions about the effect of one variable on another are valid, and not confounded by other variables. In experimental studies, systematic differences in characteristics can be dealt with by randomisation at the stage of data collection. Characteristics will be randomly distributed between groups, such that observed differences at the end of the study can be attributed to the effect of the experimental intervention. In cohort studies, where randomisation is clearly not possible, a method for dealing with such characteristics during the stage of data analysis can be used; the effect of one variable on another can be measured while simultaneously adjusting for the effects of other characteristics, for example using multiple linear regression. It is, however, unlikely that all of the variables which are thought to confound the relationship of interest have in fact been measured, and therefore they cannot be adjusted for in the data analysis. It is also likely, that

there are variables that confound the relationship, but are not known by the researcher to do so, and have similarly not been measured in the study.

A clear advantage of fixed-effects models in analysing longitudinal data, is that there is no need to include variables in the analysis that do not (or are it is assumed that they do not) change over time within individuals, such as sex, ethnicity, age of onset of offending, as they do not contribute to the analysis. Such stable characteristics which have been considered to be relevant to the likelihood of violence may be assumed to have the same effect on the alcohol-violence relationship of that individual at each time point.

In fixed-effects models, every individual acts as his or her own control such that, in the case of the relationship between alcohol consumption and violence, the relationship between level of alcohol consumption on violence is compared at each time point; the level of violence associated with a given level of alcohol consumption at one time point is compared with the level of violence associated with a different level of alcohol consumption at every other time point for each individual. The factors that do not change over time (such as gender, ethnicity, and, within certain parameters, IQ and personality), whether they have been measured or not, do not contribute to this analysis. It is therefore the change in status within individuals (for example in individual's drinking alcohol) that is of relevance in the model. Any observed change in the outcome variable must, therefore, be due to the effects of these factors that vary over time.

This model eliminates all individuals whose status with respect to the exposure of interest does not change over time. Therefore, those whose alcohol consumption is at a constant, unchanging level do not contribute statistically to the model. Fixed effects models are therefore preferable when there is ample variation in the measurement of interest.

One of the assumptions of fixed-effects models is that the responses within people are independent of one another, but this is not necessarily so. An individual's level of alcohol consumption at one time-point, for example, is likely to be related to their alcohol consumption at another time point. Whether someone drinks alcohol at one time point is not independent of their drinking at another time-point; there is likely to be a pattern of drinking that shows continuity across time points. By assuming that the measures are independent of one another, as in the case of fixed-effects models, the resulting analyses produce standard errors that are erroneously

small, resulting in errors in the estimates of the precision of the effect size, such that the confidence intervals will be too narrow. Statistical correction for this assumption is possible, however, by calculating, what are known as robust standard errors (Robust Standard Error Estimation(Kezdi, 2004). Robust standard errors were first described by Huber and White (Huber, 1967, White, 1980) and use an alternative method for estimating standard errors using the residuals in the regression, rather than the standard methods based on maximum likelihood. An option to select analyses using robust standard errors is available within most statistical analysis packages, including Stata.

The general equation for the fixed effect model is given by:

$$Y_{it} = \ _{t} + \ x_{it} + \ z_{i} + \ _{i} + \ _{it}$$

where

Y_{it} is the outcome variable for individual, i, at time, t.

$_{t}$ is the intercept (or constant) term at time(t).

x_{it} is the time-varying independent variable such as alcohol consumption (for person i at time t).

z_{i} is the time-invariant independent variable such as sex (for person i).

and are the coefficients for x and z. The model assumes that the coefficients are time-invariant.

$_{i}$ is an error term that varies between individuals but not over time. It represents the effects of all of the time-invariant variables that have not been included in the model.

$_{it}$ is an error term that varies for each individual at each time point. It represents random variation at each time point.

In the case of analysis with only two time points, the equations for both time points are:

$$Y_{i1} = \ _1 + \ x_{i1} + \ z_i + \ _i + \ _{i1}$$

$$Y_{i2} = \ _2 + \ x_{i2} + \ z_i + \ _i + \ _{i2}$$

A "first difference" equation is then produced from subtracting the second equation from the first:

$$Y_{i2} \ \ Y_{i2} = (\ _2 \ \ \ _1) + \ (x_{i2} \ \ x_{i1}) + \ (z_i \ \ z_i) + (\ _i \ \ \ _i) + (\ _{i2} \ \ \ _{i1})$$

This can be rewritten as:

$$Y_i = \ \ + \ x_i + \ \ _i$$

Where (delta) represents a change or difference score. It is apparent that $(\ _i \ \ \ _i)$ and $(z_i \ \ z_i)$ are both equal to zero, and therefore do not contribute to the equation and are eliminated. In the analysis of the relationship between alcohol consumption and violence for a given person, for example, the difference in violence is equal to the difference in alcohol consumption, plus the difference in the constant (or intercept), plus the difference in the measured random error. All of the other terms that do not change within the individual over time, such as sex or ethnicity, clearly have a difference of zero, and are eliminated from the equation.

The above principles can be extended and applied to the analysis of data in which there are more than two time-points.

RANDOM-EFFECTS MODELS

Random effects models explicitly take into account the clustering of the data, by introducing into the standard regression equation a term that represents variation between clusters: Below is the standard equation for the linear predictor of an individual in cluster j. It can be seen that there is an error term, e_j, which represents the amount that varies between clusters, and is known as the random effect.

$$j = \quad + \quad {}_1x_1 + \quad {}_2x_2 + \quad {}_3x_3 + \ldots + \quad {}_rx_r + e_j$$

Unlike fixed-effects models, in which variation between clusters is "differenced out", random effects models explicitly include the variation between clusters, which is treated rather like any other predictor or covariate in a multiple linear regression model. As stated earlier, multiple linear regression models produce estimates of the relationship between exposures and outcome, while adjusting for all of the covariates in the model simultaneously. Random-effects models are, in essence, very similar, but they also include a term for the variation between clusters (the random effect) which, when treated as any other covariate, produces estimates of the relationship between exposures and outcome while controlling for the effect of variation between clusters. In the case of longitudinal studies, the clustering is at the individual level, and therefore it is the variation between individuals that is estimated and adjusted for. Change over time in this context is often termed- "growth", and the models that are used to describe this are often known as "growth models", but also as "hierarchical models" or "multilevel models of change". The main modification applied to the standard random-effects model in order to study growth is the introduction of an interaction term between the variation between clusters -the random effect; in my study, the random effect would be the variation between individuals and the measure of time.

It should be noted that the odds ratios derived from fixed-effects models are not interpreted in the same way as those derived in random-effect models. In fixed-effects models, the odds ratios are derived from the overall population-averaged probabilities adjusted for the covariates to derive the odds ratios. In contrast,

random-effects models produce subject-specific odds-ratios. In a random coefficient model, individual participant's starting level (intercept) is allowed to vary, and so the odds ratio derived from the model is applies to individuals who have the same starting value. The odds ratios are therefore said to be conditional on the intercept, or "subject specific", and therefore apply to other individuals with the same starting level.

CHOOSING BETWEEN RANDOM OR FIXED-EFFECTS

In contrast to fixed-effects models, random effects models provide a more precise estimate of the effect size. There are, however, more stringent assumptions of the model; all of the variables that may influence the outcome variable, including all of the relevant interactions between variables, should be specified in the model. This may, however, be a problem in that some variables may not have been measured. Lack of their inclusion may result in biased or incorrect estimates of the outcome.

If there are no omitted variables, or if the omitted variables are not correlated with variables that are in the model, a random-effects model is preferable. Random-effects models (if correctly specified) will result in appropriately smaller standard errors than equivalent fixed-effects model. Also, random-effects models, unlike fixed-effects models allow the effect of time invariant characteristics on the dependent variable to be estimated.

The assumptions made about $_i$ ($_i$ represents all of the time-invariant characteristics that have not been included in the model) are important in determining which model, either fixed or random effects, is appropriate. If $_i$ is believed to be correlated with the time-varying independent variables in the model (the *x*s) this would violate the assumptions of the random effects model (that the time-invariant variables excluded from the model are not correlated with the time-invariant variables in the model), and a fixed effect model should be selected instead.

Another assumption of the random-effects model is that the model contains all relevant covariates, including interactions. To test whether to include a particular interaction term, a comparison of the model fit either with or without the interaction term is made using the likelihood ratio test. Such models are termed

"nested models", which means than all of the terms of the smaller model (e.g. without the interaction terms) are included in the larger model (with the interaction terms).

As stated, a problem of using the random-effects model is that the estimates may be biased due to the failure to include all relevant covariates. In the current study, the group of covariates available for inclusion in the models may not be complete, but are nevertheless comprehensive, and given the marked similarity in the estimates for alcohol consumption when both the fixed- and random- effects models were applied in preliminary analyses (the validity of the latter does not require the inclusion of all covariates), it was decided to proceed with random effects models so as to include the full dataset.

MARGINAL EFFECT

The marginal effect (also known as partial derivative or partial change) is the difference in probability of the outcome when the exposure is present versus not present. The marginal effect of x (an exposure) is therefore the difference in the probability of $y = 1$ (the outcome), given $x = 1$, minus the probability that $y = 1$ given $x = 0$).

In studies in which the effect of exposure on outcome varies according to a third variable, (for example the effect of alcohol on violence varying with age) the marginal effect of alcohol on violence can therefore be calculated (extrapolated) and plotted for different ages to aid the interpretation of estimates of change.

STRUCTURAL EQUATION MODELS

Structural equation models (SEM) can be thought of as, in principle, an extension of multiple linear regression. However, whereas in ordinary multiple regression it is possible to estimate the relationships between a single dependent variable and multiple independent variables, in SEM it is possible to simultaneously estimate a set of relationships between one or more dependent and one or more independent variables. Another advantage of SEM is that, whereas in ordinary multiple regression only relationships between measured (observed) variables can be estimated, in SEM estimation of latent variables can be modelled. Latent variables

are used to represent abstract phenomena such as a behaviour that cannot be measured directly through observation. Indirect measures of the phenomena may be obtained for example by self-report questionnaires. These measured scores are called "observed" or "manifest" variables in SEM parlance. Multiple observed variables then serve as "indicators" for the underlying latent phenomena they are presumed to measure.

The first step in SEM is to determine the statistical structure of the data using factor analysis of the observed data to determine the extent to which the observed measures are represented by underlying factor or factors. Factor analysis is a technique used to investigate whether a number of observed variables are linearly related to a smaller set of unobservable factors. It is used to identify groupings of observed variables that are correlated with each other and to create a smaller number of underlying latent variables, known as factors.

Two types of factor analyses are common in SEM; exploratory factor analysis (EFA) and confirmatory factor analysis (CFA). EFA models are used when the underlying structure of the relationship between the observed variables is not known, whereas in CFA, prior understanding of the structure of the relationship between variables will be used to specify a model. EFA could for example be applied to a new questionnaire designed to measure facets of personality to determine the extent to which the items of the questionnaire (observed variables) were related to the underlying latent constructs of personality facets. The strength of the relationship of the observed variable to its latent construct is given by the "factor loading" (which can be thought of as a standardised regression coefficient), and therefore, in the case of the new questionnaire, it would be favourable that questions would show high factor loadings for a particular facet they were supposed to measure, and low or negative loadings for other facets. CFA could be used with an established validated questionnaire to confirm that the given questions are related to the assumed underlying construct in a given dataset. The goals of CFA modelling are therefore to confirm the hypothesized structure of the data, but may also be used to test competing hypotheses.

Once the model has been specified (the relationship between the observed variables and the underlying factors), the "model fit" is then evaluated. This is a measure of how well the model as specified explains the data. SEM software generates several "goodness-of-fit" statistics to estimate the extent to which the

model “fits” the data. Although there is no agreement on the use of any single goodness-of-fit statistic, the ones most commonly used are the Chi-Squared Test of Model fit, Comparative Fit Index (CFI), Tucker-Lewis Fit Index (TLI) and Akaike’s Information Criterion (AIC)(Byrne, 2012).

Alternative models should be specified as there may be multiple alternative models that “fit” the data, and the most appropriate should be selected using both model fit statistics and hypothesis to guide the choice. Once, the most appropriate model has been specified and model fit has been established, the model can then be specified to investigate linear relationships between factors.

CHAPTER 8 STUDY METHODS

DESCRIPTION OF STUDY

My research is based on a longitudinal cohort study using data from the National Longitudinal Study of Adolescent Health (Add Health). Add Health is a study of nationally representative adolescents in the United States of America (USA), which commenced in 1994-95. Data collection interviews took place in four waves, the most recent in 2008.

The study is coordinated by the University of North Carolina. Anonymised data were made available for this study by way of contract and data use agreement between the University of Cardiff and the University of North Carolina.

WAVE I SAMPLING METHODS

Participants were selected from 80 high schools (9^{th}-12^{th} grade) and 52 middle schools 6^{th}-8^{th} grade) with unequal probability of selection (the probability was proportional to the size of each school). The school was the primary sampling unit (PSU). A school was included in the randomisation if it had more than 30 students and an 11th grade (typically the penultimate year of secondary school education and accommodating students aged 16-17). For each high school selected, one of its feeder schools (usually a middle school) was selected with a probability proportional to its student contribution to the high school. This yielded one school pair in each of 80 different communities. If one of the originally selected schools did not agree to participate in the study, replacement schools were selected within each stratum until an eligible school was found. More than 70% of the originally selected schools agreed to participate in the study. The study design ensured that the sample was representative of US schools with respect to country of origin, school size, school type, urbanicity, and ethnicity.

Wave I was carried out in 2 phases: In the first phase (between September 1994 until April 1995) questionnaires were administered to over 90,000 students while at school. Each student was interviewed on a single day within one 45 to 60 minute

class period. The school interviews provided measurements of school activities, health conditions and the school context.

In the second phase, a core sample of students was then selected to take part in a 90-minute interview, which took part in the student's home. Approximately 500 researchers were trained to undertake these interviews across the country. Interviews were carried out with the written consent of both the adolescent and a parent or guardian. To form the sample, students were stratified by school grade and sex. Approximately 17 students were selected randomly from each strata in each school pair. This provided the "*core sample*".

In addition, four specific samples were recruited using a different sampling strategy. This was done to provide sufficiently large samples for specific analyses. These samples were:

The saturation sample. This included all students from 16 of the selected schools (2 large schools and 14 small schools).

The disabled sample. Eligibility was determined by response to questions on disability status.

Ethnic samples. Individuals who were from so-called high education Black families (defined as originating from families in which at least one parent had a college degree), Cuban, Puerto Rican or Chinese ethnicities.

Genetic samples. Identical and fraternal twins, full siblings, half siblings and unrelated adolescent pairs in the same home were selected based on responses in the in-school questionnaire.

The latter three groups were selected on response to questions in the in-school questionnaire.

The core sample and selected samples together made a total of 20,745 adolescents in wave 1, representing 79% of sampled students. This sample formed the basis for all subsequent follow-up interviews and for the basis of this study.

Audio-computer assisted self interview (ACASI) on laptop computers was used for sensitive health and health risk behaviour questions in all waves. This technology has been found to improve the quality of self reporting sensitive information (Turner *et al.*, 1998a).

Parent Questionnaire

A parent of each participant, who was preferably the mother who resided with the participant also completed an interviewer-assisted questionnaire at Wave I. The questionnaire covered topic including household income, neighbourhood characteristics, education, and characteristics of their child who was participating in the survey.

WAVE II

Approximately one year later, in 1996, all adolescents in school grades 7-11, and those in grade 12 who were part of the genetic sample, took part in a further in-home interview. 14,738 completed this interview giving a response rate for Wave II of 88.6%.

WAVE III

Wave III interviews were conducted between August 2001 and April 2002. Participants were, by then, aged between 18 and 26. A 77.4% response rate was achieved (N=15,197). Responses were recorded on laptop computers. The average length of the interview was 134 minutes. As in wave II, sensitive material was entered by the respondent in privacy.

Respondents were followed up in all geographical locations in the USA, including Alaska and Hawaii. Even those who were incarcerated were also re-interviewed.

Biological specimens were taken during wave 3 including saliva from the genetic sample for DNA extraction.

WAVE IV

The fourth wave of data collection took place in 2007-08, when the participants were between 24-32 years of age. In total, 92.5% of the sample was located; 15,701 individuals completed an in-home interview yielding a response rate of

80.3%. The interview took 90 minutes and data was collected using similar laptop-based technology as in previous waves.

SAMPLE WEIGHTS

Data were collected from children from randomly selected schools. As stated above, the school was the primary sampling unit, and therefore children were clustered within schools. In addition, several groups were deliberately oversampled in order to increase the efficiency of the study, and non all participants responded at every wave. Sample weights are then necessary to provide unbiased and nationally representative estimates. The purpose of the sample weights are therefore to 1. ensure the sample totals are unbiased estimates of population totals; 2. to adjust for differences in probability of selection across different members of the sample; 3. to adjust for differences in response rate across different subgroups of the sample, and 4. to adjust for random fluctuations of the composition of the composition of the population as a whole.

To correct for design effects, the model fit is as follows:

OUTCOME = COVARIATES + DESIGN VARIABLES + ERROR TERMS

The design variables and error terms describe the clustering, stratification, weight variables and the correlation structure of the data. The covariates are the variables of interest in the analysis. If the design variables and error terms are excluded from the analysis, the estimates of ratios, variances and standard errors will be incorrect. If weights are used but the design structure is ignored, totals and ratios will be correct, however estimates of variances and standard errors will be incorrect. It is therefore necessary to undertake a design-based analysis that takes account of the sampling structure to give unbiased estimates of variances and standard errors.

Sample weights have been calculated by the Add Health research team, and were made available for this study. An 8-step method by which the sample weights were calculated using a method know as inverse probability weighting have been described in detail (Tourangeau and Shin, 1999). Inverse probability weighting (IPW)is a technique whereby a model is generated using characteristics that are associated with dropping out of the study generate a predicted probability for each individual to remain in the study. The IPW is the inverse of the predictive

probability of remaining in the study. For example, a model is created using characteristics such as age, gender, socio-economic status (an all other factors that are associated with study drop-out. A participant who has a predictive probability of remaining in the study of only 1% is given a weight of 100, whereas an individual who has a predictive probability of remaining in the study of 50% would have a weight of 2. Wave I sample weights were calculated to take into account the complex sampling design. Although a stratification variable was not included in the initial sampling plan, a post-stratification adjustment was made by region of the country (North-East, Midwest, South, and West). Weights for Wave II, III and IV allow adjustment for the sampling design, and also non-response at preceding Waves.

CHAPTER 9 EXPOSURE AND OUTCOME VARIABLES CHOSEN TO TEST THE RELATIONSHIP BETWEEN EARLY ALCOHOL CONSUMPTION AND LATER VIOLENCE

EXPOSURE DATA

ALCOHOL USE

The main exposure of interest in this study is alcohol use. Several questions had been asked at each of the four waves regarding patterns of alcohol use and its effects.

The variables extracted for the purpose of analyses were:

Over the past 12 months on how many days did you drink 5 or more drinks in a row?

1 or 2 days
Once a month
2 or 3 days month
1 or 2 days a week
3 to 5 days a week
Every/almost every day

Over the past 12 months, on how many days have you gotten drunk or "very, very high" on alcohol ?

1 or 2 days
Once a month
2 or 3 days per month
1 or 2 days a week
3 to 5 days a week
Every/almost every day

During the past 12 months, on how many days did you drink alcohol?

1 or 2 days
Once a month
2 or 3 days per month
1 or 2 days a week
3 to 5 days a week
Every/almost every day

Think of all the times you have had a drink during the past 12 months. How many did you usually have each time? (A "drink" is a glass of wine, a can of beer, wine cooler, a shot glass of liquor, or a mixed drink.)

A variable named *total alcohol quantity* was created to capture the total quantity of alcohol ingested over the past year. This variable was coded by multiplying the variable representing the number of days alcohol was consumed by the number of drinks usually consumed on each occasion. The product was then collapsed into 6 categories for further analysis.

CIGARETTE SMOKING AND CANNABIS USE

The secondary exposures of interest in this study were cigarette smoking and cannabis use. Participants were asked the following questions:

During the past 30 days, on how many days did you smoke cigarettes?

During the past 30 days, on the days you smoked, how many cigarettes did you smoke each day?

During the past 30 days, on how many days did you smoke marijuana (cannabis)*?*

Participants entered their responses to the above questions as integers. A variable named *total cigarettes smoked* was created which was a product of these response.

OUTCOME DATA

My outcome of interest in this study is the perpetration of violence. The primary outcome of interest is the perpetration of serious violence. There are three subsidiary violence outcome measures in this study; they are frequency of fighting, frequency of fighting in group, and frequency of fighting due to drinking alcohol.

PRIMARY OUTCOME VARIABLE: SERIOUS VIOLENCE

The primary violence variable is derived from the participants' self-report of their behaviour.

I chose the question about self-reported serious violence for analysis, as it was similar across waves. The question in Wave I and II was:

In the past 12 months, how often did you hurt someone badly enough to need bandages or care from a doctor or nurse?

Never
1 or 2 times
3 or 4 times
5 or more times

In Wave III and IV the wording of the question was slightly different to that in Wave II in that the words "in a physical fight" were inserted:

In the past 12 months, how often did you hurt someone badly enough in a physical fight that he or she needed care from a doctor or nurse?

Never
1 or 2 times
3 or 4 times
5 or more times

The main outcome measure for serious violence, was dichotomised prior to analysis. Although this reduces the detail of the information available for analysis, many methods of analysis entail the assumption that the predictor and outcome variable are normally distributed, on an equal-interval scale, and linearly related to one another. In most cases, measures in psychiatry and criminology are not of this type, and there are significant advantages to dichotomizing data of this type to avoid invalidating assumptions of parametric statistical models (Farrington, 2000).

Other authors who have constructed a violence measure from Add Health data have included the questions "*How often have you pulled a knife or gun on someone*" and " how often have you shot or stabbed someone" to make a composite (Reingle *et al.*, 2012b, Reingle *et al.*, 2011). In the present study, those questions were excluded, in favour of the question used, "*How often have you hurt someone to need bandages or care from a doctor or nurse*" as it incorporates violence resulting from weapon use, yet excludes threats of violence which could be made in the furtherance of other crime such as robbery, or in self defence, in which no physical aggression took place.

SECONDARY OUTCOME VARIABLES

FIGHTING

This question was asked at wave I, II and IV and concerned self-reported physical fighting in the 12-months before interview. The question was:

How often did you get into a serious physical fight?

Never
1 or 2 times
3 or 4 times
5 or more times

GROUP FIGHTING

The frequency of self-reported fighting in a group against another group was asked at all 4 waves. The question was:

In the past 12 months, how often did you take part in a fight where a group of your friends was against another group?

Never
1 or 2 times
2 or 4 times
5 or more times

FIGHTING DUE TO DRINKING

Participants were asked how often they had fought which they attributed to drinking alcohol at waves I, II and III. The question they were asked was:

Over the past 12 months how many times did you get into a physical fight because you had been drinking?

Never
Once
Twice
3-4 times
5 or more times

POTENTIAL CONFOUNDERS

INDIVIDUAL FACTORS

AGE

Participants were required to enter their date of birth. Their age in years on the day of their participation in each of the waves in the study was calculated.

ETHNICITY

The interviewer was asked to endorse a category at wave I that best represented the participant's ethnic origin. These responses were then categorised as White, Black, Asian / Pacific Islander, or Other.

IQ

IQ was measured at wave I using The Adolescent Health Picture Vocabulary Test (AHPVT). This is an 88-item test adapted from the Peabody Picture Vocabulary Test, Revised (PPVT)(Dunn and Dunn, 1981) and a test of verbal ability, however it can be standardised and converted to an IQ score. The individual item scores were not available for analysis, however the Cronbach's alpha of the original scale has been reported to be 0.93, so I accepted the scores as reliable and valid.

DELINQUENCY

The delinquency questionnaire was administered at wave I, and comprised of the following questions:

In the past 12 months, how often did you paint graffiti or signs on someone else's property or in a public place?

In the past months, how often did you deliberately damage property that didn't belong to?

In the past 12 months, how often did you lie to your parents or guardians about where you had been all whom you were with?

How often did you take something from the store without paying for it?

How often did you run away from home?

How often did you drive a car without its owner's permission?

In the past 12 months, how often did you steal something with more than $50?

How often did you go into a house or building to steal something?

How often did you sell marijuana or other drugs?

How often did you steal something worth less than $50?

How often we allowed, rowdy, or unruly in a public place?

All questions were answered on a Likert scale with the following possible responses:

Never
1 or 2 times
3 or 4 times
5 or more times
refused/don't know/not applicable

These 11 variables showed good internal consistency with Cronbach's alpha of 0.80. Principal component factor analysis indicated that the scale is unidimensional with 92.6% of the variance explained by the 1st factor.

IMPULSIVITY

I selected five items from a set of questions about personality characteristic to represent impulsivity. The questionnaire was administered at wave I, and on the basis of face validity, the following questions were extracted to represent impulsivity. They were:

When making decisions, you usually go with your "gut feeling" without thinking too much about the consequences of each alternative?

When you have a problem to solve, one of the first things you do is get as many facts about the problems as possible?

When you are attempting to find a solution to a problem, you usually try to think of as many different ways to approach the problem as possible?

When making decisions, you usually use a systematic method for judging and comparing alternatives?

After carrying out a solution to a problem, you usually try to analyse what went right and what went wrong?

Each item was scored on a 5-point Likert scale. The scoring on item 1 was reversed so as to be consistent in direction with the other 4 items.

The item-test correlations (Cronbach's alpha) were 0.52, 0.71, 0.72, 0.70, and 0.67 respectively. The Cronbach's alpha for the combined scale with the 5 items included was 0.66. Given the relatively low item-test correlation (0.52) of item 1 (When making decisions, you usually go with your "gut feeling" without thinking too much about the consequences of each alternative?) I constructed a scale with the remaining 4 items. This resulted in higher correlations (0.72 to 0.76), with an overall alpha for the scale of 0.74.

I then carried out principal component analysis. This showed that 64% of the variance was explained by the first factor, and approximately 13% by each of two further factors. Although it would have been possible to use the factor loadings of the first principal component as the variable score instead of the sum of the individual items, it was decided that it was preferable for reasons of simplicity in interpretation of analysis to construct the scale using the sum of the individual items, given that the Cronbach's alpha demonstrated satisfactory internal consistency.

PEER SUBSTANCE USE

Participants were asked three questions relating to substance use of their peers at waves I and II. They were:

Of your three best friends, how many drink alcohol at least once a month?

Of your three best friends, how many smoke at least once cigarette a day?

Of your three best friends, how many use marijuana (cannabis) at least once a month?

For each question, subjects entered an integer between 1 and 3. These values were then added to create a scale representing peer substance use.

DEPRESSION

Subjects completed a 19-item scale at wave I that was designed to collect information about their current emotional state, particularly low mood. The items are adapted from the Center for Epidemiologic Studies of Depression Scale (CES-D) (Radloff, 1977).

The questions were:

How often was each of the following things true during the past week:
You were bothered by things that usually don't bother you
You didn't feel like eating, your appetite was poor.
You felt that you could not shake off the blues, even with help from your family and your friends.
You felt that you were just as good as other people.
You had trouble keeping your mind on what you are doing.
You felt depressed.
You felt that you were too tired to do things.
You felt hopeful about the future.
You thought your life had been a failure.
You are fearful.
You are happy.
You talked less than usual.
You felt lonely.
People were unfriendly to you.
You enjoyed life.
You felt sad.
You felt that people dislike you.
It was hard to get started doing things.
You felt life is not worth living.

Each question response was recorded on a 4-point Likert scale corresponding to:
Never or rarely.
Sometimes.
A lot of the time.
Most of the time or all the time.
Refused/don't know

The scoring of items 4, 8, 11, and 15 was reversed for consistency. The Cronbach's alpha reliability coefficient for this scale was 0.86. The total score was retained for further analysis.

TEMPER

The adult completing the parent interview at wave I was asked the following question:

Does (name of child) have a bad temper?
Yes
No

CLOSENESS TO PARENT

A 4-item scale was used as a measure of closeness to parents closeness at wave I. Participants were asked the following about each parent

How close do you feel to your (parent)?
Most of the time, your (parent) is warm and loving to you
You are satisfied with the way your parent and you communicate with each other
Overall, you are satisfied with your relationship with your parent

Item 1 was scored on a 5-point Likert scale:

Not at all
Very little
Somewhat
Quite a bit
Very much

Items 2-4 were scored on a 5-point Likert scale as follows:

Strongly agree
Agree
Neither agree nor disagree
Disagree
Strongly disagree

If the participant did not have a parent figure, s/he was allocated the lowest score in the scale. The questions showed good internal consistency, with Cronbach's alphas of 0.84 and 0.88 for the scales for maternal and paternal closeness respectively.

FAMILY CHARACTERISTICS

FAMILY STRUCTURE

During the in-home interviews at wave I, participants were asked to name every member of their household. In turn, the relationship of each household member to the participant was then ascertained (e.g. mother, father, sister, stepmother). I created a variable was to represent the structure of the family as follows:

"*Conventional"* family composition was defined as a household including the participants' biological mother and father.

A "*parent-partner"* family was defined as a household including either a biological father or mother and a parental partner, or foster parents, or adoptive parents.

"*Single parent family*" was defined as one in which the participant lived with either his/her biological mother or biological father, but no other parent figure.

Each category held, with or without other family members living there.

SIZE OF HOUSEHOLD

Participants were asked the question at wave I:

How many people are in your household?

The number reported was used in analysis.

PARENTAL SUPERVISION

Direct parent supervision was measured using a scale constructed from 3 items ascertained at wave I. All participants were asked the following 3 questions about their mother and father if resident:

How often is (your parent) at home when you leave school?
How often is (your parent) at home when you return from school?
How often is (your parent) at home when you go to bed?

Answers were recorded on a 5-point Likert scale:

Never
Almost never
Some of the time
Most of the time
Always

The total score of the 3 items was used to construct the index. As there were responses for both resident mother and resident father, the highest score was taken. These scales had Cronbach's alpha of 0.54 and 0.77 respectively. It can be seen that the individual items to not measure the same thing, as being at home when the child leaves for school is not the same as being at home when they return from school or go to bed. The total of these items together given an indication of the amount of supervision that a child has, although can not measure the quality of the supervision.

PARENTAL CONFLICT

The participant's parent who completed the parent interview at wave I was asked:

How much do you fight or argue with your current (spouse/partner)?

Not at all
A little
Some
A lot

I gave those parents who did not have a partner the code "not at all". This was because for this study I considered the level of conflict at home that the individual witnessed to be important. If all those who had only one parent at home were coded as "missing" on this variable, too much data would have been lost.

NEIGHBOURHOOD FACTORS

Detailed contextual information about the participant's community was available from the US Census Bureau. These data consisted of nationally collected social, demographic, and criminological data, presented as an average for a defined

geographical area. The geographical areas were defined at 4 levels of decreasing size. They were the state, county, tract, and block group. The state and county levels were according to nationally defined state and county boundaries. A tract is defined by the US Census Bureau as "*A small locally defined statistical area within selected counties, generally having stable boundaries and, when first established by local committees, designed to have relatively homogenous demographic characteristics. They do not cross county boundaries*". Tracts contain between 2,000 and 8,000 people, with an average population of 4,000. A census block is defined as "*A small, usually compact area, bounded by streets and other prominent physical features as well as certain legal boundaries*". Blocks do not cross census tract or county boundaries. A block group is a cluster of census blocks, and averages 452 housing units or 1,100 people (1990)

I used census block data in my analyses.

NEIGHBOURHOOD DISADVANTAGE

The following variables were selected as indicators of neighbourhood disadvantage at census block level. They were:

Proportion of low birth weight births per all births
Proportion of single parent households
Proportion of households with income less than $15,000 Per anum
Proportion of persons aged over 25 without high school diploma or equivalent
Unemployment rate.

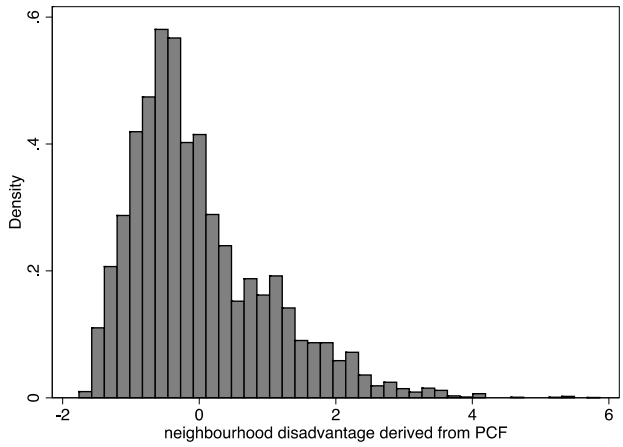
For parsimony, a scale was constructed to represent overall neighbourhood disadvantage. Unlike the construction of a scale by combining similar items with comparable response ranges, in keeping with similar methods used in a previous study (Vazspnyi *et al.*, 2006), the scale was constructed using factor analysis. Principal component factor analysis, with oblique rotation, to allow for correlations between variables, was therefore used to derive an appropriate scale. The variable representing the proportion of low birth weight did not load on to the 1st factor, and the factor loading for the variable representing the proportion of low birth weight births was modest (0.47). This variable was therefore dropped. Principal component analysis using the remaining 4 variables showed that the items loaded

strongly onto a single factor. The factor loadings were 0.76, 0.88, 0.81 and 0.83 with respect to the proportion of single parent families, the proportion of households with income less than $15,000, the proportion of people with high school diploma, and unemployment rate respectively.

I used these factor loadings to create a neighbourhood disadvantage score. High scores indicated higher neighbourhood disadvantage.

The resulting neighbourhood disadvantage variable had a mean of 0 and a standard deviation of 1 (range of -1.8 to 5.9). A histogram of the scores in this constructed scale is shown in Figure 1.

Figure 1. Distribution of scores on constructed neighbourhood disadvantage scale



NEIGHBOURHOOD VIOLENT CRIME RATE

The violent crime rate per hundred thousand of the population was available at the level of each census block group. The variable was divided into three categories (tertiles) to represent low, medium and high violent crime rates.

POPULATION DENSITY

The population density at each Census Block Group was available, and was divided into three equal categories, representing low, medium and high for further analysis.

PERSONALITY DIMENSIONS

Personality traits were assessed at Wave IV using the Mini-IPIP. This is a short form of the 60 item International Personality Item Pool-Five-Factor model measure (Goldberg, 1999), which has retains just four items for each of the 'big five' traits. It has sound psychometrics (Donnellan *et al.*, 2006), to measure the "Big-Five" personality traits (extraversion, neuroticism, agreeableness, openness and conscientiousness) with the instructions, "*How much do you agree with the each statement about you as you generally are now, not how you would like to be in the future"* . Each statement required a response on a 5-point likert scale:

Strongly agree
Agree
Neither agree nor disagree
Disagree
Strongly disagree

Questions within the scale were then aggregated to form subscales representing 5 dimensions of personality. The questions used in the construction of each subscale are shown below. Some items had their scores reversed before combining to form a subscale

EXTRAVERSION

I am the life of the party (reversed)
I talk a lot
I talk to a lot of different people at parties (reversed)
I keep in the background

NEUROTICISM

I have frequent mood swings (reversed)
I am relaxed most of the time
I get upset easily
I seldom feel blue

AGREEABLENESS

I sympathize with others' feelings (reversed)
I am not interested in other people's problems
I feel other's emotions (reversed)
I am not really interested in others

OPENNESS TO EXPERIENCE

I have a vivid imagination (reversed)
I am not really interested in abstract ideas

I have difficulty understanding abstract ideas
I do not have a good imagination

CONSCIENTIOUSNESS

I get chores done right away (reversed)
I often forget to put things back in their proper place
I like order (reversed)
I make a mess of things

ANGRY-HOSTILITY SCALE

An anger-hostility-aggression scale was administered which containing 4 items derived from items within the anger facet of the NEO PI-R (Costa and McCrae, 1992) , which is a 240-item questionnaire measuring the five personality traits of the Five Factor Model and the 6 subordinate facets. The items used in the current study were:

I get angry easily (scores reversed)
I rarely get irritated
I lose my temper (scores reversed)
I keep my cool

CHAPTER 10 STATISTICAL ANALYSIS

I used Stata 12.0 SE for all data analysis(StataCorp, 2012), except for structural equation modelling, which was carried out using Mplus Verision 7 (Muthen and Muthen, 1998-2012).

FREQUENCIES

Participants who contributed to more than one wave were included in the analyses. Thus a variable was created for each participant, which was the total number of waves for which they contributed data; those who contributed to less than 2 waves were therefore removed.

Frequencies for all exposure and outcome variables were obtained from the dataset. Weighted proportions were calculated using methods known as "*complex survey methods*" in Stata 12 (StataCorp, 2012). These methods take into account the study design, including the non-random sampling design, stratification, and participant attrition so that the estimates are generalisable to the national population. Based on these features, each participant was allocated a sample weight, and hence may contribute proportionately differing amounts to the analyses. The sample weights were included in the Add Health dataset.

Frequencies and weighted proportions were estimated and tabulated by wave of data collection, and estimated proportions (with 95% confidence intervals for the estimates in the case of line graphs) were represented graphically for selected exposure and outcome variables for ease of interpretation.

ASSOCIATION BETWEEN ALCOHOL, CIGARETTE SMOKING, OR CANNABIS USE AND VIOLENCE

The first section of analyses concerned the association between exposures and incident (new onset) violence after wave I. Crude (unadjusted) associations between exposure and outcome were first calculated using a chi-squared test, with

probability of rejecting the null hypothesis expressed at the 95% level of confidence.

The analysis of the association between substance use and incident violence was carried out using "complex survey methods" in Stata (StataCorp, 2012). The relationship between alcohol, cannabis use and cigarette smoking and incident violence was therefore initially investigated using logistic regression with complex survey methods.

The extent to which either frequency or quantity of alcohol consumption may be important in the relationship with violence was investigated. First, the correlation between frequency and amount of alcohol consumed was computed, and the model was tested for multicollinearity by calculating the variance inflation factors (VIF). Collinearity between two factors implies that the two variables are linear combinations of one another. When two factors that are collinear are entered into a regression model, the estimates of the coefficients cannot be uniquely computed. A VIF above 10 indicates likely collinearity. In the current model, there was a correlation of 0.78 between the two variables. The VIF was below 5 indicating that that the alcohol quantity and frequency of alcohol consumption variables are not collinear, and may be entered simultaneously into a model.

The number needed to prevent violence was calculated using the relevant odds ratio and the patient expected event rate (PEER) (Section IChapter 7 Epidemiological Methodology).

The analyses were then extended to investigate the effect of exposure of alcohol, on subsequent incident violence across the next 4 waves of data collection. The initial method chosen was generalised estimating equation (GEE) modelling to fit logistic regression models. GEE is a type of population average approach, which means that it gives an average change in the population mean of the outcome of interest for a given unit of change in the exposure across all people observed. The main advantage of the GEE approach is that it adjusts for multiple observations of individuals, and therefore takes account of the fact that the multiple observations of the same individual are not independent of one another. It is necessary to specify the appropriate working correlation structure of the data (the main ones are termed either independent, exchangeable or first-order auto-regressive). Failure to specify the correct correlation structure can result in inaccurate standard errors of the estimates (Pan and Connett, 2002). In practice, particularly when

there are is a large number of participants and few waves of data, (as in the present study) there is very little difference in the results whichever correlation structure is selected. The methods were then repeated to investigate the relationship between cigarette smoking and cannabis use on violence.

Random-effects models were then applied to investigate the relationship between either alcohol cigarette smoking and cannabis use and violence in the entire cohort, taking into account changes in levels of both exposure and outcome at each wave of data collection, and adjusting for assumed static confounders. To aid interpretation, the marginal effects were then calculated and plotted to cover the period between early adolescence and early adulthood.

RELATIONSHIP BETWEEN ALCOHOL, PERSONALITY FACTORS AND VIOLENCE

Finally, structural equation modelling in Mplus version 7 (Muthen and Muthen, 1998-2012) was used to investigate the relationship between alcohol and personality factors. The unadjusted associations between personality factors and violence were first calculated. Age was considered to be continuous and normally distributed. All other manifest variables were defined as ordered categorical. Univariate descriptive statistics were first generated on the study population using Stata version 12. Mplus version 7 was used for structural equation modelling in a two-step process. Confirmatory factor analyses (CFA) were carried out to determine the adequacy of the factor loadings, model fit, and correlations of the latent factors. A confirmatory factor analysis was carried out to determine whether each of the variables loaded onto their respective latent constructs. All of the factors (extraversion, agreeableness, conscientiousness, neuroticism, openness, anger-hostility, alcohol and violence) were each indicated by 4 variables (see Chapter 9). In the initial analysis, all factors loadings were allowed to vary freely except for the first measure of each factor, which was constrained at 1.0 to identify the metric of the latent variable. All factor intercorrelations were freed, as were the error terms within the same measure.

Structural models were then tested to evaluate the total, direct and indirect relationships between alcohol, violence and personality traits using age in years as a covariate. I used WLSMV parameter estimation (Weighted Least Squares Means

and Variance estimation) to correct for multivariate non-normality. Mplus allows multivariate modelling using all available data to account for missing data, which was employed in the current study. Bootstrapping with 1000 draws was used to calculate confidence intervals of the standardised path estimates.

A confirmatory factor analysis was carried out to confirm that the variables loaded on to their respective latent constructs.

Evaluation of model fit was guided by reference to the following indices: Comparative Fit Index (CFI) (Bentler, 1990), Tucker-Lewis Fit Index (TLI) (Tucker and Lewis, 1973), Root Mean Square Error of Approximation (RMSEA)(Steiger and Lind, 1980), and the chi-squared test. Accepted fit criteria are accepted to be for both CFI and TFI >0.95, RMSEA <0.06-0.08, and a non significant chi-squared test.

SECTION III RESULTS

The results are set out in four section: first the descriptive statistics of the cohort are described, secondly the investigation of the relationship between each of the substances alcohol, cigarette smoking and cannabis use and incident violence, thirdly an analyses of the relationship between the substances and violence over time among the whole cohort, and fourth a cross-sectional analysis of the relationship between personality factors and alcohol misuse on violence.

CHAPTER 11 DESCRIPTIVE STATISTICS OF ADD HEALTH

AGE AND SEX DISTRIBUTION OF THE COHORT

20,743 young people were interviewed at wave I, but only those who were in school grades 7-11, (age 12-17) or who were in school grade 12 (age 17-18) and part of the genetic or adopted sample, were followed up at wave II. These 17,519 (84.5%) participants from the initial cohort formed my sample; these participants were allocated a study sample weight because of the special characteristics that qualified them for repeated interviews. The median age of the cohort included in my sample was 15.89 years (range 11.39 to 21.24) at inception. There were 8,748 males (49.93%) and 8771 females (50.07%).

FOLLOW-UP RATE

14,100 individuals (80% of the cohort used in my study) were re-interviewed at wave II, approximately 1 year after wave I (median of 338 days); 12,991 (74.2%) were re-interviewed at wave III, a median of 6.33 years after wave I (range 5.56 years – 8.01 years, which included 382 participants who were interviewed at waves I and III, but not at wave II. Finally, 9,421 (72% of wave I) participants were re-interviewed at wave IV, a median of 12.85 years after wave I (range 11.56 years to 14.50 years).

ALCOHOL USE DATA

FREQUENCY OF ALCOHOL USE

17,519 participants provided a valid response to questions about how often they drank alcohol, with missing data on only 36 individuals (0.21%). Table 4 shows the frequencies and weighted proportions of those endorsing each response (the weighted proportion is that calculated incorporating sample weights, using complex survey methods in Stata, to account for the unequal probability of selection of each individual, and, in waves II to IV, and to adjust for sample attrition), and thus give estimates of the rates within the national population of the USA.

At Wave I (median age 15.9) 7720 (47%) of individuals reported drinking alcohol on at least one day in the previous year (see Table 4). This proportion was rather similar to 45% (6098) at Wave II, but increased to around 74% (around 9450) at waves III and IV. As expected, between waves I and II on the one hand and waves III and IV on the other, there was also an overall increase in the proportions of people drinking regularly; the proportion of people drinking 2 or 3 days a month doubles from around 8% to 16%, and those drinking 3 to 5 times per week more than trebles from 2.6% to over 8%.

Table 4. Frequency of alcohol use: reported number of days alcohol was consumed over the year prior to interviews at Wave I - Wave IV

How many days drank alcohol in last year	Frequency (Weighted %)			
	Wave I	Wave II	Wave III	Wave IV
None	9,799 (52.9%)	7,969 (55.2%)	3,702 (27.0%)	3,720 (25.8%)
1 or 2 days	2,916 (16.8%)	1,921 (13.5%)	1,473 (10.9%)	1,416 (10.3%)
Once a month	1,998 (12.1%)	1,642 (12.2%)	2,117 (16.2%)	2,010 (15.2%)
2 or 3 days per month	1,262 (8.0%)	1,087 (8.4%)	2,085 (15.8%)	2,163 (16.7%)
1 or 2 days a week	970 (6.5%)	968 (7.1%)	2,359 (19.9%)	2,564 (20.3%)
3 to 5 days a week	538 (2.6%)	351 (2.6%)	918 (8.0%)	1,097 (9.1%)
Every / almost every day	112 (1.1%)	138 (1.0%)	273 (2.3%)	362 (2.7%)
Total	17,519	14,067	12,927	13,332

NUMBER OF DRINKS CONSUMED ON EACH OCCASION

There were 17,394 people who responded to this question; of those, 9,852 (56.6%) were categorised as having not consumed alcohol (comprised of those that legitimately skipped this question having responded that they do not drink alcohol to an earlier question, combined with those that reported drinking no alcohol on each "*drinking occasion*"). There were 5,664 people who reported consuming one or more drink on each occasion (42.8% of those who responded). Of those who drank alcohol, the median number of drinks consumed at each time at wave I was 3, interquartile range 2-6. The median number of drinks consumed was also 3 at wave II and IV, but was 4 (IQR 2-7) at wave II (see Table 5).

Table 5. Median number of drinks usually consumed on each occasion among those who drank alcohol (wave I - wave IV)

	Wave I n=7,542	Wave II n=5,951	Wave III n=9,138	Wave IV n=9,510
Number of drinks usually consumed Median (IQR)	3 (2-6)	4 (2-7)	3 (2-6)	3 (2-5)

FREQUENCY OF BINGE DRINKING

At wave I, 17,479 participants provided an answer to the question: "*Over the past 12 months, on how many days did you drink five or more drinks in a row?*" Those who drank five or more drinks in a row on at least one or two occasions were classified as having participated in "*binge drinking*". Approximately one quarter reported binge drinking at wave I according to this definition, but by waves III and IV, nearly half of respondents reported binge drinking at least once. The proportion of those who reported binge drinking between 2 or 3 days a month and 1 or 2 days a week approximately doubled from around 5% in waves I and II and 10% in waves III and IV. Approximately 1% of people reported binge drinking every day or nearly every day at each of the 4 waves.

The responses to the question relating to binge drinking were cross-checked against those given for the number of drinks consumed on each occasion for reliability. At wave I, a total of 16.0% of individuals reported that they usually

drank 5 or more drinks each occasion. There were 353 people, representing 2.03% of the total that gave responses to questions that they usually drank at least 5 drinks each occasion, but reported no days of binge drinking (defined as drinking 5 or more drinks on one occasion) in the last 12 months. Although these responses are incompatible with each other, all responses were retained for analysis. Similar discrepancies were found at each of the other waves. An error rate of 2% was considered acceptable for these analyses.

Table 6. Reported number of binge drinking days* in the 12 months prior to interview at each of the waves I-IV

Number of days drank 5 or more drinks in a row in last year	Frequency (Weighted %)			
	Wave I	Wave II	Wave III	Wave IV
None	13,271 (72.9%)	10,244 (70.9%)	6,844 (48.7%)	7,038 (48.8%)
1 or 2 days	1,499 (9.2%)	1,251 (9.3%)	2,041 (15.7%)	2,172 (17.7%)
Once a month	956 (5.9%)	871 (6.7%)	1,292 (11.3%)	1,430 (11.5%)
2 or 3 days per month	712 (4.8%)	661 (5.3%)	1,103 (9.4%)	1,149 (9.7%)
1 or 2 days a week	623 (4.5%)	622 (4.5%)	1,155 (10.5%)	997 (8.2%)
3 to 5 days a week	273 (2.0%)	276 (2.3%)	404 (3.7%)	381 (3.1%)
Every / almost every day	145 (0.9%)	150 (1.1%)	94 (0.7%)	130 (1.0%)
Total	17,479	14,055	12,927	13,332

*binge drinking was defined as consuming five or more drinks in a row on a drinking day

FREQUENCY OF ALCOHOL INTOXICATION

Participants were asked to quantify how often in the past 12 months they were "*very drunk or very high*" on alcohol. As with the other alcohol variables, the patterns of responses were similar between waves I and II and between waves III and IV, but differed between I and II on the one hand and III and IV on the other (see Table 7). The responses were again cross-checked for accuracy. There were 277 (2.12%) responses in which the frequency of binge drinking was given as greater than the frequency of drinking occasions. As with previous variables, the

frequency of incompatible responses was considered low, and all subjects were retained in the analyses.

As expected, the frequency of intoxication increased over time. Approximately 30% of the sample reported getting "*very drunk or very high*" on alcohol at least once in the past year at waves I and II; this increased to nearly 50% in waves III and IV.

Table 7. Reported number of intoxicated days* reported for the 12 months prior to interview in each wave (I-IV)

Number of days intoxicated in last year	Frequency (Weighted %)			
	Wave I	Wave II	Wave III	Wave IV
None	12,973 (71.0%)	10,082 (69.6%)	6,540 (47.0%)	6,914 (48.1%)
1 or 2 days	1,957 (12.0%)	1,548 (11.5%)	2,407 (18.6%)	2,911 (22.8%)
Once a month	970 (6.4%)	892 (6.8%)	1,564 (13.3%)	1,572 (12.8%)
2 or 3 days per month	729 (4.8%)	626 (5.0%)	1,156 (10.0%)	946 (7.7%)
1 or 2 days a week	536 (3.7%)	573 (4.3%)	958 (8.8%)	705 (6.3%)
3 to 5 days a week	208 (1.6%)	223 (1.8%)	242 (2.0%)	208 (1.8%)
Every / almost every day	115 (0.6%)	129 (1.1%)	57 (0.4%)	53 (0.4%)
Total	17,483	14,055	12,927	13,332

*being intoxicated was defined as being very drunk or very high on alcohol, according to self-report

TOTAL QUANTITY OF ALCOHOL CONSUMED

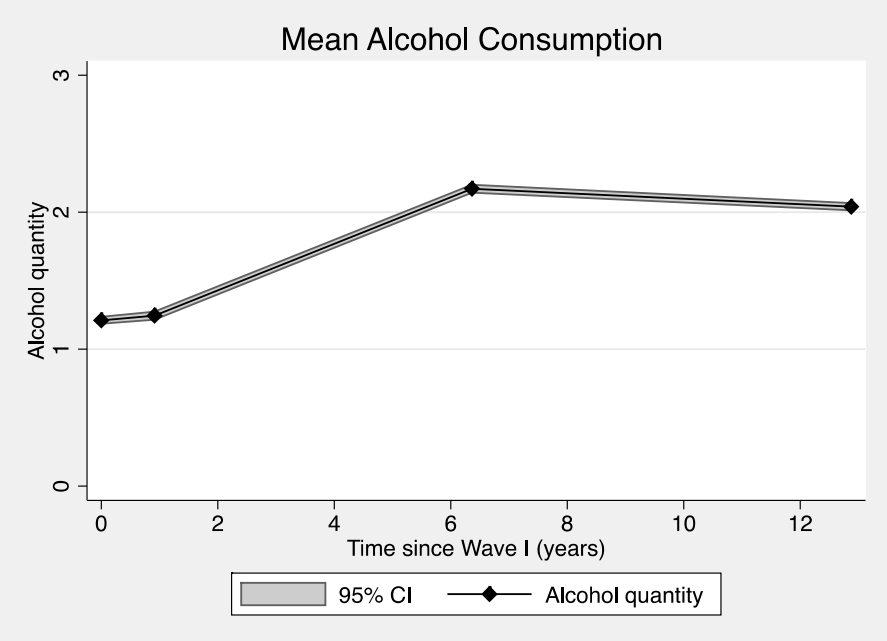
A variable was created to represent the total quantity of alcohol consumed by multiplying the category representing the number of days that alcohol was consumed by the number of drinks consumed on each occasion. The product was then divided into 6 categories for further analysis to aid interpretation. At every wave, the largest category comprised participants who did not consume alcohol. Table 8 shows that in the first two waves, just over half of the cohort had effectively described themselves as non-drinkers, but in both the second two waves, this had fallen to just over a quarter. There was an insignificant fall in the next, small category of light drinkers, but all other categories except the highest followed a

pattern of higher alcohol consumption reported in the second two waves than in the first two. Figure 2 shows the mean (and 95% confidence interval of the mean) of the total amount of alcohol consumed as defined at each wave.

Table 8. Self-reported total quantity of alcohol consumed in the 12-months prior to interview in each wave of the study

Total quantity of alcohol consumed	Frequency (Weighted %)			
	Wave I	Wave II	Wave III	Wave IV
0 (No alcohol)	9,816 (53.4%)	7,696 (55.5%)	3,702 (27.6%)	3,720 (26.3%)
1	2,156 (12.1%)	1,274 (8.9%)	1,194 (8.0%)	1,308 (9.1%)
2	1,818 (10.8%)	1,378 (9.7%)	2,192 (15.9%)	2,567 (18.5%)
3	1,365 (8.6%)	1,151 (8.8%)	2,282 (17.9%)	2,721 (21.5%)
4	1,240 (8.6%)	1,213 (9.4%)	2,140 (19.1%)	2,009 (17.5%)
5 (highest)	963 (6.6%)	935 (7.4%)	1,330 (11.5%)	901 (7.1%)
Total				

Figure 2. Mean total alcohol consumption, waves I-IV

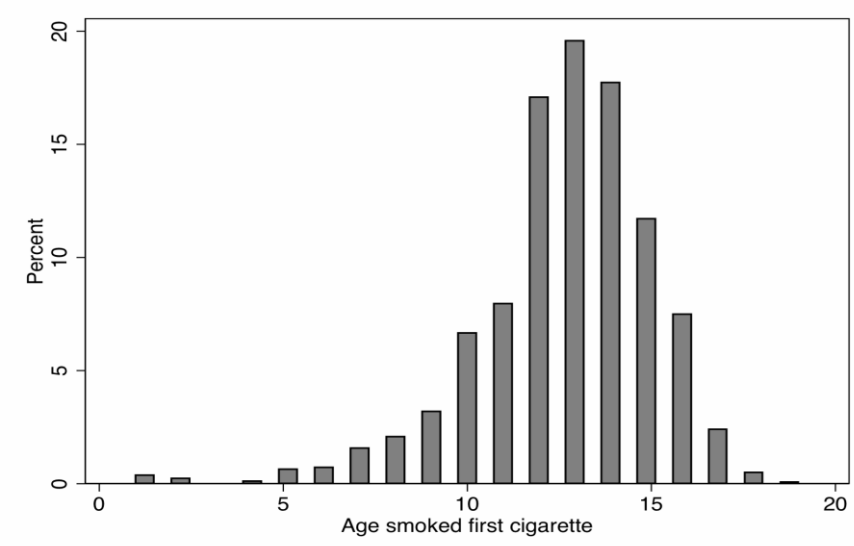


CIGARETTE SMOKING

At wave I, there was missing data for only 123 (0.7%) individuals for the number of cigarettes they smoked. At waves II-IV, there was missing data for 76 (0.54%), 42(0.32%) and 118 (0.88%) respectively.

Of the 7,140 of the participants (weighted proportion 42.8% of 17,428 valid responses) said that they had ever smoked at least one cigarette. The mean age of smoking the first cigarette was 12.7 years (SD 2.49). The mean age of smoking first cigarette was approximately normally distributed, as show in Figure 3.

Figure 3. The age at which respondents said they first smoked cigarettes (among those that had ever smoked by the time of wave I interviews)



Participants were asked on how many days they had smoked cigarettes during the 30 days prior to the first interview. Of those who had smoked, 1,403 (34.3%) had smoked daily. The median number of days which cigarettes were smoked was 15 (IQR 3-30). The median number of cigarettes smoked on each occasion was 3 (IQR 1-8).

A variable representing the total number of cigarettes smoked in the 30 days prior to interview was created by multiplying the number of days on which cigarettes were smoked, by the mean number of cigarettes smoked per day. The number of cigarettes smoked prior to wave I is shown in Figure 4. The number of days cigarettes smoked, and the estimated total number of cigarettes smoked in the 30 days prior to interview at each wave is shown in Table 9.

Figure 4. Total number of cigarettes smoked in the 30 days prior to wave I interviews among those that had smoked at least one cigarette

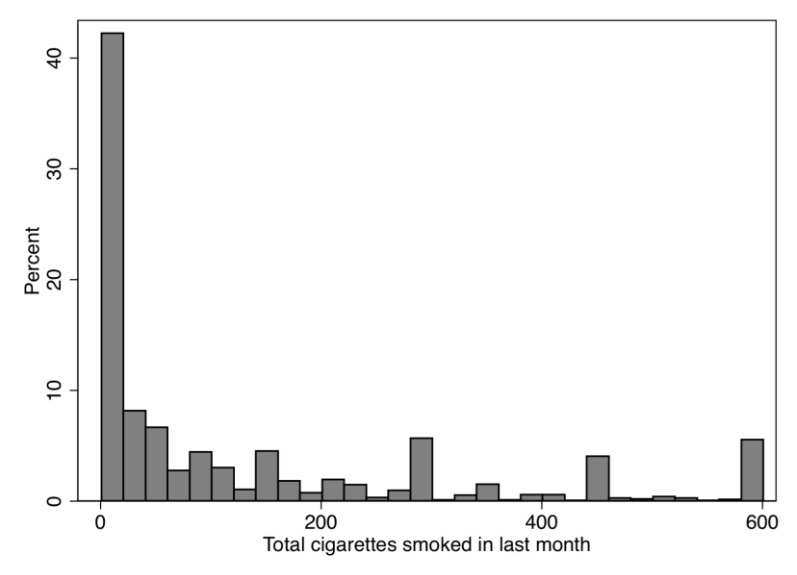


Table 9. Self-reported smoking habits of participants in the 30 days prior to interviews at waves I-IV

	Frequency (Weighted %) Wave I	Wave II	Wave III	Wave IV
Number who smoked at least one cigarette in last 30 days	4,314 (25.9)	4,407 (34.3)	4,218 (34.8)	4,925 (37.9)
Median number of days smoked at least one cigarette in last 30 among smokers	15 IQR (3-30)	15 IQR (3-30)	30 IQR (20-30)	30 IQR (12-30)
Median number of cigarettes smoked in last 30 days among smokers	49 IQR (5-216)	50 IQR (5-290)	300 (87-480)	210 IQR (40-450)
Total	17,396	14,024	12,949	13,237

CANNABIS USE

At wave I 330 (1.89%) did not give a response to the questions about their cannabis use. At waves II-IV there was missing data for 231 (1.63%), 46 (0.35%) and 17 (0.17%) of individuals. 2,345 (13.3%) of the participants reported that they had used cannabis at all in the 30 days prior to their wave I interview. The median number of times used was 3 (IQR 1-12). Approximately 95% of the people who used cannabis did so 30 or fewer times in the previous 30 days. Figure 5 (censored at 30 uses in the period), shows the distribution of responses among those who had ever used cannabis. Table 10 shows the number of people who used cannabis, and the median number of times used in the 30 days prior to each interview.

Figure 5. Number of times cannabis used in the 30 days prior to interview at Wave I among those who used it at least once (censored at 30 times)

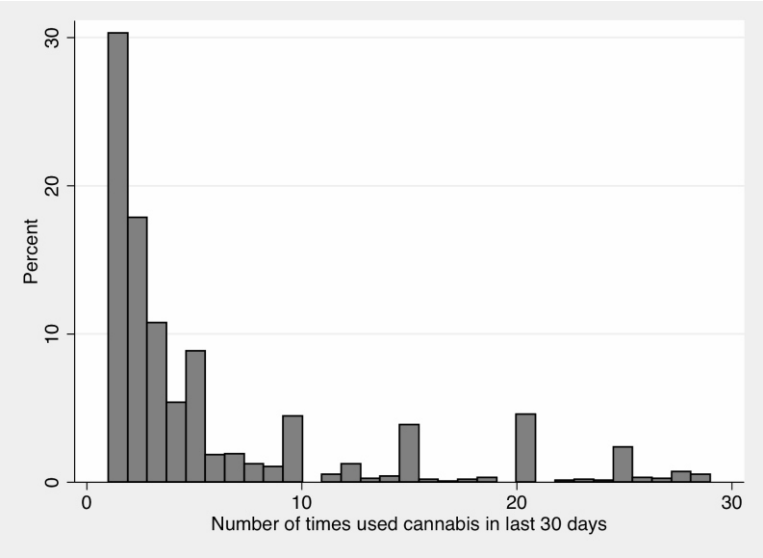


Table 10. Self-reported cannabis use of participants in the 30 days prior to interview at each wave

	Frequency (Weighted %) Wave I	Wave II	Wave III	Wave IV
Number who used cannabis at least once in last 30 days	2,345 (13.3)	2,179 (16.2)	2,848 (24.1)	2,192 (17.8)
Median number of times used cannabis in last 30 days among cannabis users	3 IQR (1-12)	4 IQR (2-14)	6 IQR (2-25)	8 IQR (2-30)
Total	17,189	13,869	12,945	13,338

VIOLENCE DATA

NUMBER COLLECTED AND MISSING DATA

Almost 20% of the 17,363 responders reported violence to another person, serious enough for their victim to need treatment, in the year prior to the wave I interview. Less than 1% (156, 0.89%) of cases were missing for this item. There were missing data on alcohol use in 0.95% of individuals who reported new onset violence at Wave II (95% CI 0.94%-2.40%), and in 0.12% of individuals who reported no new

violence at wave II (95% CI 0.10%-0.24%). The difference in proportion was statistically significant (z=4.20, p<0.001). Of those reporting new onset violence at wave III there was no missing alcohol data, however there was missing alcohol data in 0.14% of subjects (95% CI 0.07% - 0.24) who reported no new onset of violence. At wave IV, there was no missing data on alcohol use among those that reported new onset violence, and there was missing data on alcohol use 0.14% (95% CI 0.07-0.27%) of those that reported no new onset violence.

The frequency of reported serious violence in each wave is shown in Table 11. At wave II, the proportion had fallen to just over 8%, and it fell again at waves III (6.4%) and again at wave IV (2.1%). The proportions were significantly different from each wave to the next (p<0.0001). The prevalence of violence at each wave is shown in Figure 6, and shows that there is a reduction in the prevalence of violence across waves, with the sharpest reduction between wave I and wave II.

Table 11. Frequency of serious violence by wave of data collection

	Frequency (Weighted %)			
Number of times hurt someone badly enough to need bandages or care from a doctor or nurse	Wave I	Wave II	Wave III	Wave IV
0 (Never)	13,951 (80.9%)	12,931 (91.7%)	12,020 (93.6%)	13,084 (97.9%)
1 (1 or 2 times)	2,593 (14.8%)	916 (6.8%)	621 (5.2%)	238 (1.9%)
2 (3 or 4 times)	441 (2.3%)	151 (1.0%)	80 (0.7%)	22 (0.2%)
3 (5 or more times)	378 (2.1%)	80 (0.5%)	64 (0.5%)	9 (0.06%)
Total	17,363	14,078	12,785	13,353

INCIDENT VIOLENCE

Table 13 gives a summary of the reporting patterns of incident violence among those who provided data at all 4 waves. Most sustained their non-violence, a small minority (5 people) were consistently violent, and the rest showed the full range of possible patterns between them. The patterns of responses are very similar among this group when compared to those who were not violent at wave I. There was a slightly lower proportion of people who reported no violence at any of waves II-IV

among those who reported no violence at wave I compared to those who reported violence at wave I (90.4% compared with 92.3%); this was a significant reduction ($p<0.0001$).

As shown in Table 13 there were 7,208 people who were not violent at any wave, compared with 600 (7.7%) who reported violence on at least one later wave of data collection (incident violence)[1]. A total of 275 (3.5%) individuals reported violence for the first time at wave II, 274 (3.5%) at wave III and 51 (0.7%) at wave IV.

Table 12. Frequency of serious incident violence by wave of data collection

	Frequency (Weighted %)		
Number of times hurt someone badly enough to need bandages or care from a doctor or nurse	Wave II	Wave III	Wave IV
Never	10,879 (95.8%)	10,879 (96.1%)	11,904 (93.4%)
1 or 2 times	389 (3.7%)	389 (3.4%)	701 (5.5%)
3 or 4 times	38 (3.1%)	38 (0.3%)	82 (0.6%)
5 or more times	19 (0.17%)	19 (0.2%)	56 (0.4%)
Total	11,325	11,325	12,743

[1] Those who were not violent at wave I were defined as a "subpopulation" of the entire cohort. By specifying the subpopulation, only those who comprise the subpopulation are included in the calculation of the estimate, but all cases are used to calculate the standard errors. This technique ensures that the standard errors of the estimates are calculated correctly, and that the estimates remain representative of the national population.

Table 13. Patters of responses of those reporting serious incident violence at least once in last 12 months in waves II-IV

Serious violence at least once in last 12 months among those who reported no violence at wave I	Wave II	Wave III	Wave IV	Frequency (%)
	No	No	No	7208 (92.3)
	No	No	Yes	51 (0.7)
	No	Yes	No	257 (3.3)
	Yes	No	No	219 (2.8)
	No	Yes	Yes	17 (0.2)
	Yes	No	Yes	5 (0.1)
	Yes	Yes	No	46 (0.6)
	Yes	Yes	Yes	5 (0.1)

SECONDARY MEASURES OF VIOLENCE

Three additional measures of violence were analysed; they were: serious physical fighting, group fighting, and fighting due to alcohol. The frequencies of responses for each measure at each time point are shown below.

SERIOUS PHYSICAL FIGHTING

Participants were asked how many times they had been in a serious physical fight in the 12-months before interview. The results are shown in Table 14 below. Approximately one third of participants reported that they had been in at least one serious physical fight in the past 12 months. The frequency reduced to 20% and 5% at waves II and IV respectively. (This question was not asked at wave III).

Table 14. Frequency of serious physical fighting by wave of data collection

	Frequency (Weighted %)		
Number of times got into a serious physical fight in last 12 months	Wave I	Wave II	Wave IV
0 (Never)	11,577 (66.4%)	11,266 (80.3%)	12,604 (94.5%)
1 (1 or 2 times)	4,065 (23.8%)	2,318 (16.5%)	633 (5.0%)
2 (3 or 4 times)	928 (5.4%)	290 (2.1%)	50 (0.04%)
3 (5 or more times)	797 (4.5%)	163 (1.2%)	32 (0.02%)
Total	15,579	14,037	13,319

GROUP VIOLENCE

How often participants had taken part in a fight, one group against another in the 12 months before interview was asked at each wave. Approximately 21% had taken part in such a fight at least once in the 12 months before interview at wave I and wave II. The frequency fell to approximately 9% and 3% at waves III and IV (see Table 15).

Table 15. Frequency of group violence by wave of data collection

	Frequency (Weighted %)			
Number of took part in a fight, one group against another in past 12 months	Wave I	Wave II	Wave III	Wave IV
0 (Never)	13,772 (79.3%)	11,493 (81.3%)	11,734 (89.8%)	12,870 (96.5%)
1 (1 or 2 times)	2,371 (16.0%)	1,937 (14.1%)	920 (8.2%)	392 (3.1%)
2 (3 or 4 times)	485 (2.5%)	349 (2.7%)	149 (1.5%)	44 (0.3%)
3 (5 or more times)	387 (2.2%)	255 (0.2%)	72 (0.1%)	15 (0.1%)
Total	17,375	14,034	12,875	13,321

FIGHTING DUE TO ALCOHOL

At waves I, II and III, participants were asked how often they had been in a physical fight due to drinking alcohol in the 12 months prior to interview. As shown in Table 16, approximately 6% stated they had done so at least once at both waves II and II; the proportion rose to 9% at wave III. The question was not asked at wave IV.

Of interest, compared with the responses in Table 11, there was a reduction in the proportion who reported committing serious violence at each wave, but the proportion who reported fighting attributed to alcohol slightly increased. At wave I, almost 20% of individuals stated they had been involved in perpetrating serious violence, yet only 6% stated they had been in a physical fight due to alcohol. At wave II, nearly 8% stated they had committed serious violence and 6% said they had been involved in a physical fight, which they attributed to alcohol. At wave III, approximately 6% reported committing serious violence, and 9% stated they had been involved in fighting which they attributed to alcohol.

Table 16. Frequency of physical fighting attributed to alcohol by wave of data collection

	Frequency (Weighted %)		
Number of times got into a physical fight due to drinking alcohol in last 12 months	Wave I	Wave II	Wave III
0 (Never)	16,455 (93.9%)	13,243 (94.0%)	11,965 (91.2%)
1 (Once)	647 (3.7%)	525 (3.7%)	594 (5.2%)
2 (Twice)	220 (1.4%)	166 (1.2%)	227 (2.1%)
3 (3-4 times)	112 (0.6%)	90 (0.6%)	116 (1.0%)
4 (5 or more times)	73 (0.5%)	66 (0.5)	51 (0.6)
Total	17,507	14,090	12,953

RELATIONSHIP BETWEEN PRIMARY AND SECONDARY MEASURES OF VIOLENCE

The prevalence of each of the measures of violence at every wave can be seen in Figure 6. All of the measures of violence reduced over time, except for fighting due to alcohol, which showed a slight increase. The highest prevalence was serious fighting at wave I. The reduction in prevalence in serious violence and serious fighting was in parallel, and sharpest between wave I and II.

The tetrachoric correlations between the primary and secondary measures of violence are shown in Table 17. At waves II and IV there was a perfect correlation

between serious fighting and serious violence (serious fighting was not measured at wave III). There were moderately high correlations (0.6 – 0.7) between reported serious violence and group fighting at each wave. Correlations between alcohol related fighting and the other measures of violence were low (approx. 0.55) except with group violence at wave III (0.72).

Figure 6. Prevalence of Primary and Secondary Measures of Violence by Wave

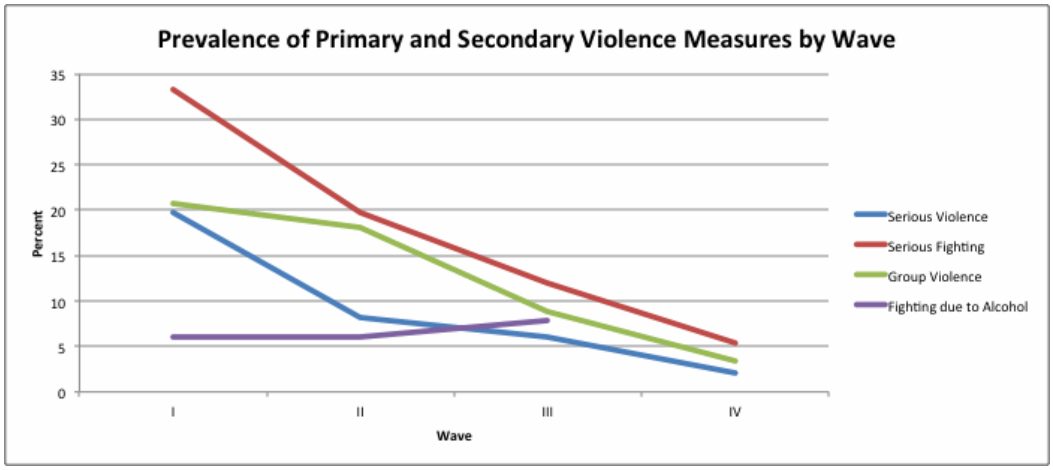


Table 17. Correlations Between Measures of Violence

	Serious Violence	Serious Fighting	Group Violence	Alcohol Fighting
	Wave I			
Serious Violence	1			
Serious Fighting	0.75	1		
Group Violence	0.60	0.64	1	
Alcohol Fighting	0.53	0.56	0.57	1
	Wave II			
Serious Violence	1			
Serious Fighting	1	1		
Group Violence	0.71	0.71	1	
Alcohol Fighting	0.57	0.56	0.53	1
	Wave III			
Serious Violence	1			
Group Violence	0.69	.	1	
Alcohol Fighting	0.56	.	0.72	1
	Wave IV			
Serious Violence	1			
Serious Fighting	1	1		
Group Violence	0.80	0.84	1	

CHAPTER 12 THE RELATIONSHIP BETWEEN ALCOHOL USE, CIGARETTE SMOKING, CANNABIS USE AND INCIDENT VIOLENCE

INTRODUCTION

In this section I explore the longitudinal relationship between alcohol, cigarette smoking and cannabis, separately and together, and incident violence. In order to obtain a "pure" effect of alcohol, cigarette smoking or cannabis use on violence, those who were already violent at wave I were excluded from the analyses. I begin with an analysis of the crude (unadjusted) relationship between any reported alcohol consumption measured at wave I and incident serious physical violence to others at wave II or later. I follow this with an exploration of the effect of potential confounders, and an estimate of the relationship between each substance and violence while adjusting for potential confounders. This is followed with a more detailed investigation using the various measures of alcohol consumption (frequency, quantity, total consumption, frequency of binge drinking, and frequency of perceived intoxication) and incident violence, while adjusting for important confounders. The relationship between cigarette smoking, and then cannabis use and violence is each then explored in the same way.

I conclude by estimating the number of people who would need to stop drinking, smoking or using cannabis to prevent one episode of violence (the number needed to prevent).

CRUDE RELATIONSHIP BETWEEN ALCOHOL USE AND INCIDENT VIOLENCE

Table 18 shows that a significantly higher proportion of young people who had been drinkers but non-violent at wave I reported violence on at least one subsequent wave of data collection (409, 6.9%) than those individuals who had reported neither drinking nor violence at wave I.

Table 18. Crude (unadjusted relationship between alcohol consumption at wave I and incident violence at waves II-IV

	Drank Alcohol at Wave I					
	No		Yes		Significance	
	N	%	N	%	X^2 (df)	p
Incident Violence after Wave I						
No	9929	94.6	5526	93.1		
Yes	561	5.4	409	6.9	16.2 (16,424)	<0.001

EXPLORATION OF POTENTIAL CONFOUNDERS

The frequency of incident violence by each individual covariate (ethnicity, gender, IQ, delinquency, depression, rational decision making, other substance) is presented in Table 19. Similarly, the frequency of incident violence by family and household factors (number of siblings, family structure, parental conflict, supervision by mother/father and closeness to mother/father) and neighbourhood factors (disadvantage, crime rate, and population density is presented in Table 20 and Table 21 respectively.

As can be seen in Table 19, all of the individual factors were strongly associated with incident violence. Impulsivity was the covariate that was most weakly associated with violence. All of the family and household factors were associated with incident violence except closeness to mother and supervision by mother. There was weak evidence that both higher neighbourhood violent crime and higher population density was associated with incident violence (p=0.06). Neighbourhood disadvantage, however, was strongly associated with violence (Chi^2 =24.68, p<0.001).

Table 19. Frequency of incident violence by confounders: individual factors

	Incident Violence					
	No		Yes		Significance	
	N	%	N	%	X^2	p
Ethnicity						
White	9907	94.6	568	5.4		
Black	3327	92.3	277	7.7		
Asian/Pacific Island	1184	96.4	44	3.6		
Other	1158	92.6	92	7.4	41.5	<0.001
Gender						
Male	6650	90.5	697	9.5		
Female	8931	96.9	286	3.1	298.5	<0.001
IQ						
Low	4594	92.7	362	7.3		
Medium	4586	94	294	6.0		
High	5618	95.3	276	4.7	33.3	<0.001
Delinquency score						
Low	4735	95.3	234	4.7		
Medium	5345	95	283	5.0		
High	5430	92.1	463	7.9	60.6	<0.001
Depression						
Low	4867	94.7	273	5.3		
Medium	5297	94.7	296	5.3		
High	5417	92.9	414	7.1	21.9	<0.001
Impulsivity						
Low	3776	94.3	230	5.7		
Medium	6539	94.6	376	5.4		
High	5266	93.3	377	6.7	9.0	0.011
Ever used cannabis						
No	11840	94.9	641	5.1		
Yes	3380	91.7	307	8.3	52.5	<0.001
Ever used cocaine						
No	15137	94.3	921	5.7		
Yes	323	88.3	43	11.7	23.4	<0.001
Ever sniffed glue						
No	14791	94.3	897	5.7		
Yes	669	90	74	10.0	23.0	<0.001
Ever used LSD						
No	14527	94.3	881	5.7		
Yes	898	91.4	84	8.6	13.4	<0.001
Temper						
No	9545	94.6	542	5.4		
Yes	3644	92	318	8.0	34.8	<0.001
Peer substance use						
None	5,078	94.8	274	5.1		
Low	5,031	93.4	375	6.6		
High	2,228	90.8	225	9.2	45.8	<0.001

Table 20. The frequency of incident violence by covariates: family factors

	Incident Violence					
	No		Yes		Significance	
	N	%	N	%	X^2	p
Number of siblings						
0-1	9204	94.5	535	5.5		
2-4	6040	93.5	423	6.5		
>4	337	93.1	25	6.9	8.32	0.02
Parents fight or argue						
Not at all	4741	93.1	350	6.9		
A little	5273	94.7	294	5.3		
Some	2692	94	172	6		
A lot	309	91.7	28	8.3	14.8	0.002
Family structure						
Both biological parents	8190	95	430	5		
Mother/Father + partner	2388	92.7	188	7.3		
Single parent	3512	92.8	273	7.2	33.4	<0.001
Supervision by Mother						
Low	3734	94.6	215	5.4		
Medium	6345	93.7	426	6.3		
High	5462	94.1	340	5.9	3.3	0.19
Supervision by Father						
Low	4682	94.5	271	5.5		
Medium	5515	94.3	335	5.7		
High	5345	93.4	376	6.6	6.51	0.04
Closeness to Mother						
High	448	93.7	30	6.3		
Medium	7231	94.1	456	5.9		
Low	7002	94.2	433	5.8		
Mother Absent	863	93.2	63	6.8	1.51	0.68
Closeness to father						
High	3645	94.1	227	5.9		
Medium	3595	95	188	5		
Low	3882	94.2	239	5.8		
Father Absent	4428	93.1	328	6.9	4.35	0.002

Table 21. Frequency of incident violence by covariates: neighbourhood factors

	Incident Violence					
	No		Yes		Significance	
	N	%	N	%	X^2	p
Violent Crime Rate						
Low	5081	94.6	290	5.4		
Medium	5048	94.0	322	6.0		
High	5005	93.5	348	6.5	5.81	0.06
Population Density						
Low	5298	94.4	312	5.6		
Medium	5163	94.3	314	5.7		
High	4973	93.4	350	6.6	5.69	0.06
Neighbourhood disadvantage						
Low	5243	94.9	283	5.1		
Medium	5102	94.5	299	5.5		
High	4791	92.7	376	7.3	24.68	<0.001

The extent to which each variable confounded the relationship between alcohol and violence was then tested by calculating the percentage change in odds ratio between alcohol use and violence after entering each variable in turn (see Table 22 and Figure 7).

As there was some variation in the number of people who provided responses to the questions, the crude and adjusted odds ratios were calculated on exactly the same sample for each variable. As can be seen both in Table 22 and Figure 7, adjusting for age resulted in the largest change in effect size, an increase in the estimated effect size of approximately 84% from 1.31 to 1.64. In other words, age is confounding (masking part of the effect) between alcohol and violence. Adjustment for peer substance use resulted in a decrease in the magnitude of the association between alcohol use and violence by 76%, from 1.33 to 1.07. Adjustment for delinquency score resulted in a decrease of effect size of approximately 71%, from 1.31 to 1.08. This means delinquency explains part of the association between alcohol and violence (higher delinquency is associated with more drinking and more violence). Other covariates had a more modest effect.

In subsequent analyses I included all covariates that, when added to the regression resulted in an adjusted odds ratio that was greater or less by more than 5% of the unadjusted odds ratio. These variables were age, sex, ethnicity, IQ, depression,

impulsivity, temper, cigarette smoking, cannabis use, neighbourhood disadvantage and neighbourhood violent crime. This approach was chosen rather than including all covariates for two reasons. First, because cases in which there is missing data on any covariate are necessarily removed in regression analyses, there is a smaller sample available for analysis. Second, the more covariates that are in a logistic regression model, the higher the standard errors. This would increase the chance of Type 2 errors (failing to reject a false null hypothesis).

Figure 7. Effect of adjustment for potential confounders in relationship between alcohol consumption and violence

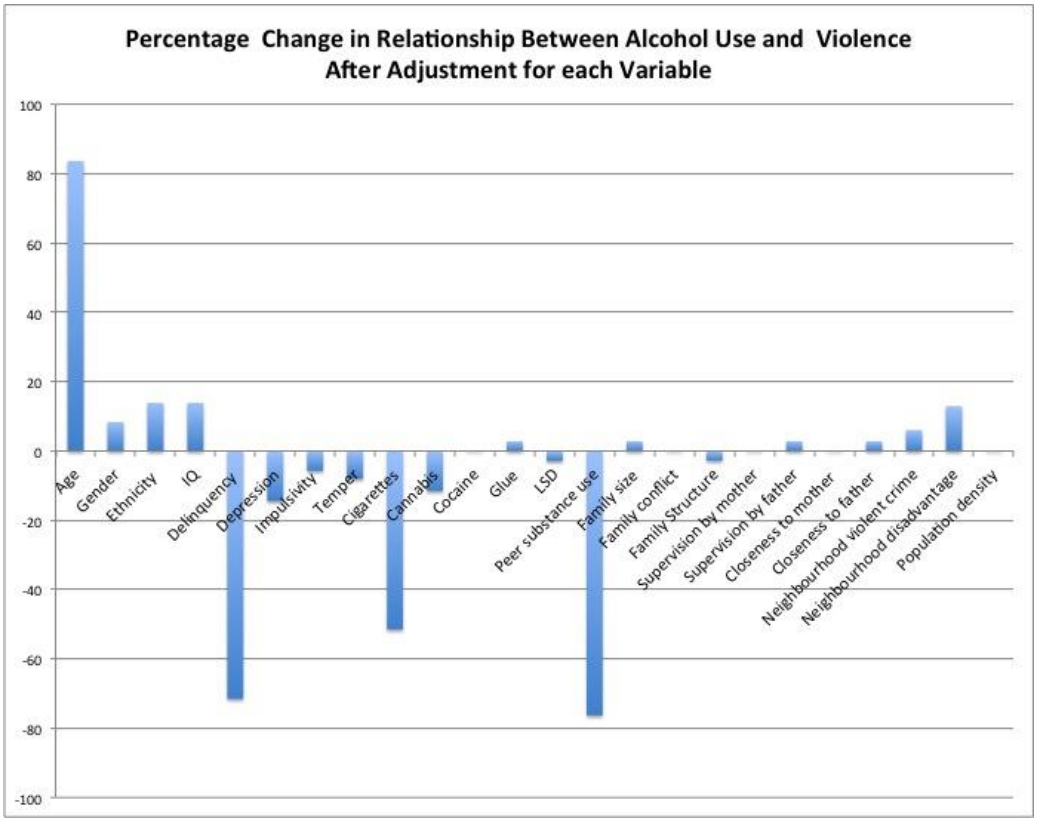


Table 22. Crude and adjusted odds ratios for covariates in relationship between total alcohol quantity at wave I and incident violence reported in waves II-IV

Covariate	n	Crude	Adjusted	Change after adjustment (%)
Individual factors				
Alcohol Quantity (6 categories)	16,425	1.31 (1.15-1.49)		
Age at Wave I.	16,421	1.31 (1.15-1.49)	1.64 (1.43-1.90)	83.7
Gender	16,425	1.31 (1.15-1.49)	1.34 (1.17-1.53)	8.4
Ethnicity	16,418	1.31 (1.15-1.49)	1.36 (1.20-1.56)	13.9
IQ	15,593	1.31 (1.14-1.49)	1.36 (1.19-1.56)	13.9
Delinquency	16,356	1.31 (1.14-1.49)	1.08 (0.94-1.25)	-71.5
Depression	16,426	1.31 (1.15-1.49)	1.26 (1.10-1.44)	-14.4
Impulsivity	16,425	1.31 (1.16-1.53)	1.29 (1.12-1.49)	-5.7
Temper	13,938	1.33 (1.15-1.49)	1.30 (1.12-1.49)	-8.0
Cigarette smoking	16,346	1.31 (1.15-1.49)	1.14 (0.98-1.32)	-51.5
Cannabis	16,078	1.32 (1.16-1.51)	1.27 (1.11-1.46)	-11.5
Cocaine	16,303	1.31 (1.14-1.49)	1.31 (1.15-1.50)	0
Glue	16,309	1.31 (1.15-1.49)	1.32 (1.15-1.50)	2.8
LSD	16,272	1.31 (1.15-1.50)	1.30 (1.14-1.49)	-2.8
Peer substance use	16,035	1.33 (1.17-1.52)	1.07 (0.91-.26)	-76.0
Family / household factors				
Family size	16,425	1.31 (1.15-1.49)	1.32 (1.16-1.51)	2.8
Family conflict	13,748	1.35 (1.17-1.55)	1.35 (1.18-1.56)	0
Family Structure	14,859	1.32 (1.16-1.52)	1.31 (1.14-1.50)	-2.7
Supervision by mother	16,384	1.31 (1.15-1.49)	1.31 (1.15-1.49)	0
Supervision by father	16,385	1.31 (1.15-1.50)	1.32 (1.16-1.51)	2.8
Closeness to mother	16,388	1.31 (1.15-1.49)	1.31 (1.15-1.50)	0
Closeness to father	16,394	1.31 (1.15-1.49)	1.32 (1.15-1.50)	2.8
Neighbourhood factors				
Neighbourhood violent crime	15,962	1.29 (1.13-1.48)	1.31 (1.15-1.50)	6.0
Neighbourhood disadvantage	15,962	1.33 (1.16-1.52)	1.38 (1.20-1.57)	13.0
Population density	16,272	1.31 (1.15-1.50)	1.31 (1.14-1.49)	0

RELATIONSHIP BETWEEN ALCOHOL USE AT WAVE I AND INCIDENT VIOLENCE AT WAVE II

The relationship between alcohol use at wave I and incident violence at wave II was calculated using logistic regression with complex survey methods. This relationship between exposure at wave I and outcome at wave II is of importance for three reasons, firstly due to the short amount of time between the two waves (less than 12 months on average); second that the effects of exposure on violence would be expected to be proximal, and third that the question about violence covered the previous 12 months and thus covered the entire follow-up period.

Rather than excluding all those who were violent at wave I by case deletion, the analysis was carried out by specifying the subgroup of those who were not violent at wave I to ensure correct estimates of standard errors. The weighted proportions are therefore included in the table (instead of absolute numbers in the sample). Weighted proportions are estimates of proportions in the national population rather than the actual proportions in the sample used for analysis.

Table 23 shows the crude and adjusted odds ratios, 95% confidence intervals and p-values for the association between alcohol use at wave I and incident violence at Wave II. The analyses show that there was a significant crude relationship between each measure of alcohol consumption and later onset of violence. Only quantity of alcohol consumed each occasion, and frequency of binge drinking remained significant after controlling for selected confounders.

Those who reported drinking between 5 and 10 drinks on each occasion were more than twice as likely to report having been violent one year later than those who reported that they did not drink alcohol (OR 2.07). The rate was even higher for those who reported drinking 11 or more drinks (OR 3.10). In fact, there was a significant linear trend - the higher the quantity of alcohol consumed on each drinking occasion, the greater the likelihood of later onset of violence (OR 1.45). Binge drinking was also significantly associated with violence. There was also a significant linear trend between frequency of binge drinking and violence. In addition, there was weak evidence of a linear relationship between frequency of getting very drunk and violence (OR 1.14, 95% 1.0-1.29, p=0.045).

Table 23. Logistic regression models of relationship between alcohol use at wave I and violence at wave II

Alcohol use	Weighted Proportion of subjects violent at Wave II (%) N=10,352	Logistic regression Odds ratio (95% CI) Crude	p	Adjusted	p
Overall quantity of alcohol consumed					
0 (none)	3.08	1		1	
1	4.22	1.39 (0.90-2.13)		1.56 (0.91-2.67)	
2	4.81	1.59 (0.99-2.54)		1.52 (0.85-2.72)	
3	6.74	2.27 (1.52-3.39)		2.10 (1.21-3.66)	
4	5.67	1.89 (1.18-3.01)		1.54 (0.75-3.18)	
5 (highest)	10.27	3.60 (2.29-5.65)		1.73 (0.85-3.48)	
Linear trend		1.26 (1.16-1.36)	<0.001	1.12 (1.00-1.27)	0.059
Number of drinks usually consumed each occasion					
0	3.07	1		1	
1-4	4.26	1.40 (1.00-1.98)		1.44 (0.91-2.30)	
5-10	7.41	2.53 (1.77-3.62)		2.07 (1.11-3.87)	
11 or more	11.42	4.07 (2.46-6.74)		3.10 (1.59-6.02)	
Linear trend		1.59 (1.38-1.83)	<0.001	1.45 (1.16-1.81)	0.001
Frequency of drinking alcohol in last year					
Never	3.08	1		1	
1 or 2 days in past year	5.00	1.65 (1.17-2.32)		1.81 (1.16-2.80)	
Once a month or less	6.31	2.12 (1.49-3.01)		2.00 (1.23-3.26)	
2 or 3 days a month	5.20	1.72 (1.05-2.83)		1.20 (0.64-2.26)	
1 or 2 days a week	8.58	2.95 (1.68-5.17)		1.45 (0.59-3.58)	
3 + days per week	5.10	1.69 (0.85-3.36)		0.49 (0.18-1.37)	
Linear trend		1.23 (1.13-1.35)	<0.001	1.03 (0.91-1.17)	0.657
Frequency of binge drinking in last year					
Never	3.31	1		1	
1 or 2 days in past year	5.71	1.77 (1.14-2.73)		1.97 (1.25-3.08)	
Once a month or less	8.01	2.54 (1.69-3.81)		2.18 (1.31-3.61)	
2 or 3 days a month	8.07	2.56 (1.49-4.40)		1.48 (0.78-2.80)	
1 or 2 days a week	7.74	2.44 (1.30-4.61)		1.57 (0.71-3.43)	
3 or more days per week	10.51	3.43 (1.91-6.16)		1.70 (0.63-4.58)	
Linear trend		1.31 (1.21-1.42)	<0.001	1.13 (1.02-1.26)	0.02
Frequency of getting very drunk in last year					
Never	3.32	1		1	
1 or 2 days in past year	6.46	2.01 (1.41-2.88)		1.67 (1.09-2.55)	
Once a month or less	6.58	2.05 (1.30-3.21)		1.99 (1.05-3.75)	
2 or 3 days a month	6.80	2.12 (1.21-3.72)		1.95 (1.07-3.58)	
1 or 2 days a week	8.71	2.78 (1.48-5.21)		2.25 (1.09-4.64)	
3 or more days per week	6.90	2.16 (0.98-4.77)		0.49 (0.12-2.03)	
Linear trend		1.27 (1.17-1.39)	<0.001	1.14 (1.0-1.29)	0.045

*Adjusted for age, delinquency, gender, ethnicity, IQ, depression, impulsivity, temper, cigarette smoking, cannabis use, peer substance use, neighbourhood disadvantage and neighbourhood violent crime Wave I. Calculated using Wave II sample weights on subgroup of individuals who were not violent at Wave I.

**Calculated as a product of the number of times drank alcohol in the month prior to interview and the average number of drinks consumed each occasion. Divided into 6 categories for analysis.

RELATIONSHIP BETWEEN ALCOHOL USE AT WAVE I AND INCIDENT VIOLENCE AT WAVES II-IV

The relationship between alcohol use at wave I and incident violence at any point after wave I (violence reported at waves II, III or IV) was then analysed. A binary variable was created such that a single episode of violence to another person, which resulted in his/her needing treatment at any of waves II to IV was coded as incident violence. These analyses were carried out using the wave IV sample weight. 7,680 participants (of 9,333) formed the subgroup of individuals who were non-violent at wave I but had valid violence data at subsequent waves, and a wave IV sample weight.

Unlike the results for violence up to wave II, there was a significant relationship between overall alcohol consumption and any subsequent violence in waves II-IV (see Table 24). Compared with those who did not drink, those in the lowest alcohol consumption category were 80% more likely to report subsequent violence, (OR 1.81, 95% CI 1.23-2.68), and those in the highest alcohol category were twice as likely to report later violence (95% CI 1.06-3.88), this being strong evidence of association. A similar picture was found in the relationships between the number of drinks usually consumed on each occasion and later violence. Although there was evidence of a positive linear association between the number of drinks usually consumed and later violence, in fact the greatest increase in risk was found in those who reported drinking between 5-10 drinks on each occasion (OR 2.53, 95% CI 1.57-4.08). There was weak evidence that individuals who reported drinking 11 or more drinks on each occasion were more likely to report subsequent violence that non-drinkers (OR 1.91, 95% CI 0.97-3.79). The analysis of the frequency of alcohol consumption revealed a significant positive association with violence

A similar pattern was observed with frequency of binge drinking, and frequency of getting very drunk. There was evidence that binge drinking once a month or less was associated with almost twice (OR 1.93) the rate of subsequent onset of violence. Those who reported getting very drunk were significantly more likely to report subsequent violence, and in fact there was strong evidence of a linear relationship between frequency of getting very drunk and violence (OR 1.14).

Table 24. Logistic regression models of the relationship between alcohol use at wave I and onset of violence reported at wave II-IV

Alcohol use	Proportion of subjects violent after Wave I (%) N=7,680	Logistic regression Odds ratio (95% CI) Crude	p	Adjusted*	p
Overall quantity of alcohol consumed					
0 (none)	6.6	1		1	
1	9.0	1.39 (1.00-1.95)		1.81 (1.23-2.68)	
2	9.3	1.44 (0.98-2.13)		1.56 (0.91-2.64)	
3	13.7	2.24 (1.56-3.20)		2.46 (1.51-4.00)	
4	10.1	1.58 (1.01-2.46)		1.61 (0.91-2.82)	
5 (highest)	13.3	2.17(1.38-3.41)		2.03 (1.06-3.88)	
Linear trend		1.17 (1.10-1.26)	<0.001	1.16 (1.05-1.29)	0.005
Number of drinks usually consumed each occasion					
0	6.6	1		1	
1-4	9.1	1.41 (1.08-1.84)		1.67 (1.20-2.34)	
5-10	14.2	2.33 (1.62-3.36)		2.53 (1.57-4.08)	
11 or more	11.5	1.83 (1.08-3.11)		1.91 (0.97-3.79)	
Linear trend		1.37 (1.21-1.56)	<0.001	1.40 (1.16-1.67)	<0.001
Frequency of drinking alcohol in last year					
Never	6.6	1		1	
1 or 2 days in past year	10.1	1.58 (1.21-2.07)		1.98 (1.40-2.80)	
Once a month or less	9.8	1.53 (1.08-2.15)		1.58 (1.02-2.44)	
2 or 3 days a month	9.9	1.55 (1.01-2.38)		1.69 (0.97-2.92)	
1 or 2 days a week	15.9	2.67 (1.62-4.40)		2.53 (1.25-5.20)	
3 + days per week	9.0	1.40 (0.74-2.67)		0.77 (0.28-2.09)	
Linear Trend		1.18 (1.09-1.28)	<0.001	1.12 (1.0-1.26)	0.050
Frequency of binge drinking in last year					
Never	7.03	1		1	
1 or 2 days in past year	11.3	1.69(1.21-2.36)		1.93 (1.23-3.03)	
Once a month or less	15.1	2.35 (1.51-3.66)		1.84 (1.07-3.18)	
2 or 3 days a month	9.9	1.45 (0.82-2.58)		1.25 (0.55-2.82)	
1 or 2 days a week	11.7	1.75 (0.95-3.21)		1.71 (0.88-3.30)	
3 or more days per week	19.1	3.12 (1.45-6.71)		1.42 (0.42-4.90)	
Linear Trend		1.24 (1.13-1.35)	<0.001	1.12 (1.00-1.27)	0.064
Frequency of getting very drunk in last year					
Never	7.3	1		1	
1 or 2 days in past year	9.6	1.35 (0.94-1.91)		1.66 (1.08-1.53)	
Once a month or less	12.7	1.85 (1.16-2.94)		1.79 (1.04-3.72)	
2 or 3 days a month	12.8	1.87 (1.12-3.14)		1.94 (1.06-3.56)	
1 or 2 days a week	14.7	2.20 (1.28-3.76)		2.23 (1.09-4.60)	
3 or more days per week	9.3	1.30 (0.56-3.03)		0.55 (0.54-3.12)	
Linear Trend		1.20 (1.10-1.31)	<0.001	1.14 (1.01-1.30)	0.020

IS FREQUENCY OF ALCOHOL CONSUMPTION OR AMOUNT CONSUMED MORE IMPORTANT IN RISK OF VIOLENCE?

The above results, and particularly the associations with violence at wave II indicate that there is a stronger relationship between the quantity of alcohol consumed and violence than the frequency of alcohol consumption. In order to test whether frequency or quantity of alcohol consumption was more important,

variables representing both the frequency of alcohol consumption and the total amount of drinks usually consumed on each occasion were entered simultaneously into a model (including the covariates used in previous models). The results indicate that alcohol frequency was not significantly associated with violence after controlling for number of drinks consumed each drinking occasion. By contrast, there was a significant relationship between violence and number of drinks consumed after controlling for drinking frequency, age, gender, ethnicity, IQ, depression, impulsivity, temper, cigarette smoking, cannabis use, peer substance use, neighbourhood disadvantage and neighbourhood violent crime wave I. As shown in Table 25 for every drink consumed, the odds of onset of violence at wave II increased by 6% (95% CI. 1% to 11%). With respect to onset of violence at any time in waves II-IV, a similar pattern was observed; for each increment in number of drinks usually consumed there was a 4% risk of subsequent violence (95% CI 0% to 14%). There was no evidence of association with frequency of drinking and subsequent violence.

Table 25. Logistic regression models of the relationship between frequency of alcohol consumption and number of drinks consumed with subsequent onset of violence

	Violence at Wave II (n=10,728)		Violence at Waves II-IV (n=7595)	
	OR (95% CI)	p	OR (95% CI)	p
Number of drinks usually consumed each occasion (per drink)	1.06 (1.01-1.11)	0.012	1.04 (1.00-1.14)	0.027
Frequency of drinking alcohol in last year	0.92 (0.76-1.12)	0.304	0.99 (0.86-1.14)	0.907

Adjusted for age, gender, ethnicity, IQ, depression, impulsivity, temper, cigarette smoking, cannabis use, peer substance use, neighbourhood disadvantage and neighbourhood violent crime Wave I on subgroup of individuals who were not violent at Wave I.

THE RELATIONSHIP BETWEEN CIGARETTE SMOKING AND CANNABIS USE AND INCIDENT VIOLENCE

The relationship between cigarette smoking or cannabis use at wave I and incident violence at wave II, and then waves II-IV was then calculated. Binary variables were created, representing whether or not the individual reported smoking at least one cigarette, or used cannabis at least once in the 30 days prior to interview. The

relationship with incident violence was then calculated using a chi-squared test. As can be seen in Table 26 there was strong evidence of a significant relationship between each variable and violence.

Table 26. Crude (unadjusted) relationship between cigarette smoking or cannabis use at wave I and incident violence at wave II

	Incident Violence at wave II					
	No		Yes		Significance	
	No.	%	No.	%	X^2	p
Smoked cigarettes at Wave I						
No	9,214	95.7	419	4.3		
Yes	2,581	88.9	321	11.1	180.9	<0.001
Used cannabis at Wave I						
No	10,405	95.4	500	4.6		
Yes	1,272	63.7	724	36.3	1973	<0.001

Table 27. Crude (unadjusted) relationship between cigarette smoking or cannabis use at wave I and incident violence at wave II-IV

	Incident Violence at waves II-IV					
	No		Yes		Significance	
	No.	%	No.	%	X^2	p
Smoked cigarettes at Wave I						
No	10,405	95.4	500	4.6		
Yes	2,723	90.1	274	9.1	92	<0.001
Used cannabis at Wave I						
No	11,552	942	718	5.9		
Yes	12,821	93.5	889	6.5	4.46	0.035

As with the relationship between alcohol and violence, the relationship between cigarette smoking or cannabis use at wave I and incident violence at waves II-IV was adjusted for potential confounders. The crude odds ratio for cigarette smoking was 1.51 (1.38-1.73), and for cannabis use it was 2.04 (1.73-2.41). Each of the potential confounders was added in turn to a logistic regression, and the percentage change in the estimate after the variable was added was calculated. Figure 8 shows the percentage change of each of the variables for both cigarette smoking and cannabis use. The greatest change was seen after adjusting for peer substance abuse, resulting in a reduction in the estimate of the association of 17% and 39% with respect cigarette smoking and cannabis use. A large change in the

relationship was also seen after adjusting for delinquency (a reduction in the risk of subsequent onset of violence of nearly 40% in the case of cigarette smoking and nearly 20% in the case of cannabis use respectively). A large increase was observed (36% and 26%) after adjusting for age. It was decided to adjust future models for all covariates that produced at least a 5% change (plus or minus) of the unadjusted estimate. Thus age gender, ethnicity, IQ, delinquency, depression, temper, peer substance use, family structure, neighbourhood violent crime, neighbourhood disadvantage and alcohol use were included as covariates in further logistic regression models. These covariates were the same as those that caused more than a 5% change in relationship between alcohol and violence, but with the addition, family structure, and without impulsivity.

The relationship between smoking or cannabis use and incident violence at wave II was then calculated using logistic regression with complex survey methods on the subpopulation who were not violent before wave I. As can be seen in Table 28, there was a strong unadjusted relationship. There was weak evidence that smoking was associated with violence after adjusting for confounders (smoking 1-60 cigarettes was associated with odds ratio 1.65 (95% CI 1.02-2.65), and smoking 61 or more cigarettes per day associated with odds ration of 2.17 (95% CI 1.2-3.9)), but no evidence of a linear trend. There was no evidence of an association between cannabis use and violence after adjusting for confounders.

The association between these substances and incident violence between waves II and IV was then calculated, and in this case, there was no significant relationship between either cigarette smoking or cannabis use and violence after adjusting for confounders.

Figure 8. Effect of adjustment for potential confounders in relationship between smoking and cannabis use and violence

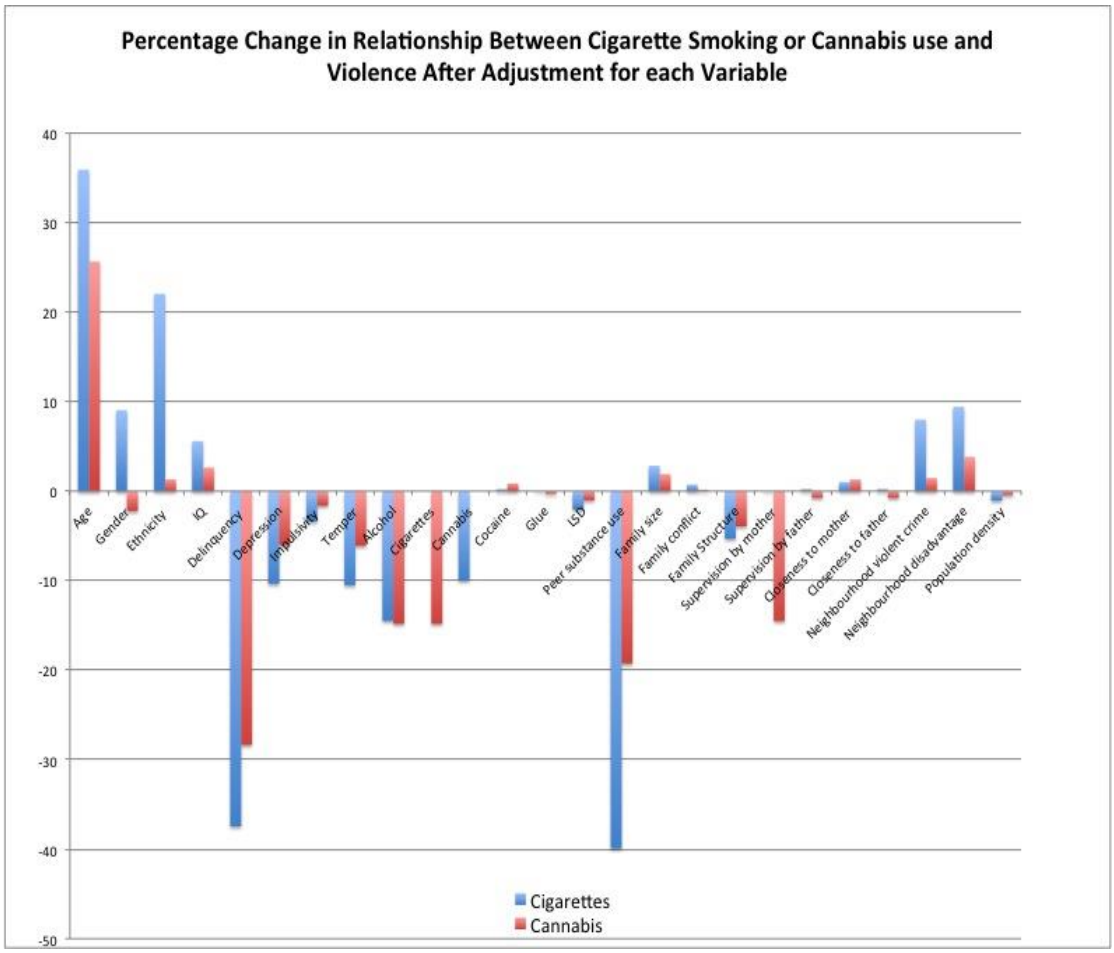


Table 28. Logistic regression models of relationship between cigarette smoking and cannabis use at wave I and violence at wave II.

	Number and weighted Proportion of subjects violent after Wave I	Logistic regression Odds ratio (95% CI)			
		Crude	p	Adjusted*	p
Total Number of times smoked cigarettes in last 30 days					
0	291 (3.3%)	1 (reference)		1 (reference)	
1-60	85 (6.4%)	2.03 (1.40-2.93)	<0.001	1.65 (1.02-2.65)	
61 or more	84 (8.4%)	2.69 (1.90-3.81)	<0.001	2.17 (1.20-3.92)	
Linear trend (per 10 cigarette)		1.014 (1.008-1.020)	<0.001	1.007 (0.997-1.018)	0.144
Number of times smoked cannabis in last 30 days					
1	355 (3.7%)	1 (reference)			
1-10	75 (7.2%)	2.12 (1.49-3.00)	<0.001	0.91 (0.53-1.57)	
11 or more	25 (8.9%)	1.19 (0.62-2.27)	0.608	0.82 (0.35-1.92)	
Linear trend (per 10 times)		1.03 (0.999-1.072)	0.055	0.979(0.823-1.164)	0.810

* Age Gender, Ethnicity, IQ, Delinquency, Depression, Temper, Family Structure, Neighbourhood Violent Crime, Neighbourhood Disadvantage, and alcohol use. Calculated using Wave IV sample weights on subgroup of individuals who were not violent at Wave I

Table 29. Logistic regression models of relationship between cigarette smoking or cannabis use at wave I and onset of violence first reported at wave II-IV

	Number and weighted Proportion of subjects violent after Wave I	Logistic regression Odds ratio (95% CI)			
		Crude	p	Adjusted*	P
Total Number of times smoked cigarettes in last 30 days					
0	425 (7.1%)	1 (reference)		1 (reference)	
1-60	153 (11.5%)	1.77 (1.29-2.41)	0.001	1.50 (1.03-2.19)	0.033
61 or more	161 (12.6%)	1.88 (1.37-2.56)	<0.001	1.61 (1.00-2.61)	0.051
Linear trend (per 10 cigarette)		1.008 (1.002-1.014)	0.014	1.00 (0.986-1.011)	0.760
Number of times smoked cannabis in last 30 days					
1	500 (7.5%)	1 (reference)			
1-10	131 (14.6%)	2.12 (1.49-3.00)	<0.001	1.30 (0.83-2.02)	0.025
11 or more	93 (8.8%)	1.29 (0.62-2.27)	0.448	0.65 (0.24-1.71)	0.377
Linear trend (per 10 times)		1.02 (0.98-1.06)	0.332	0.74 (0.55-0.99)	0.045

* Age Gender, Ethnicity, IQ, Delinquency, Depression, Temper, Family Structure, Neighbourhood Violent Crime, Neighbourhood Disadvantage, and alcohol use. Calculated using Wave IV sample weights on subgroup of individuals who were not violent at Wave I

NUMBER NEEDED TO PREVENT ONSET OF VIOLENCE

The number needed to prevent (NNP) violence was calculated for key exposures in this study. In all cases, the odds ratios and PEERs were calculated taking account of the study sampling methods by using sample weights.

Table 30 shows the NNP for five binary exposures: drank any alcohol, usually had 5 or more drinks each occasion, binge drank at least once, smoked cigarettes and used cannabis. The odds of violence one year after exposure to alcohol was about one and a half times higher than for those who were non-drinkers. Assuming that there is no residual confounding, and that the association is causative, this implies that it would be necessary for 54 people to abstain from drinking alcohol to prevent one of them from becoming violent within the following 12 months (95% CI 23-671).

In order to prevent one person from being violent, approximately 37 (range 17-189) would be required to change from usually drinking 5 or more drinks, to consuming less than 5 drinks each occasion when they drank. It would be necessary to prevent approximately 47 people (range 25-146) from binge drinking to prevent one being violent. The odds ratio for cigarette smoking was also significant; the number needed to prevent violence was 45 (12-635). Cannabis use was not associated with violence.

Table 30. Number needed to prevent one person being violence at wave II

	PEER (%)	Odds Ratio* (95% CI)	NNP (95% CI)
Drank any alcohol	3.08 (2.47-3.69)	1.63 (1.05-2.55)	54 (23-671)
Usually drank 5 or more drinks	3.44 (2.73-3.89)	1.84 (1.16-2.91)	37 (17-189)
Binge drank at least once	3.31 (2.73-3.89)	1.72 (1.23-2.41)	47 (25-146)
Smoked cigarettes	3.26 (2.64-3.89)	1.54 (1.05-2.27)	45 (12-635)
Used Cannabis	3.72 (3.18-4.25)	0.84 (0.50-1.39)	N/A

* Age Gender, Ethnicity, IQ, Delinquency, Depression, Temper, Family Conflict, Family Structure, Neighbourhood Violent Crime, Neighbourhood Disadvantage, and alcohol use. Calculated using Wave II sample weights on subgroup of individuals who were not violent at Wave I

CHAPTER 13 LONGITUDINAL RELATIONSHIP BETWEEN CHANGE IN ALCOHOL, CIGARETTE SMOKING OR CANNABIS USE AND INCIDENT VIOLENCE

INTRODUCTION

In the previous section, I showed that older age reduced the relationship between exposures to alcohol, cigarettes or cannabis and later onset of violence, suggesting that rates of violence vary with age. This was not unexpected, as I had also found in my preliminary reviews (see introduction) that rates of alcohol and other substance use vary with age and so do violence rates. In this section, therefore, I made *change the main focus of inquiry*, and in particular, the extent to which violence may change with changing substance use.

I first used a population average approach which is an extension of the analyses in the previous section, which uses a method that more formally accommodates the longitudinal design, and in particular models change in exposure over time as opposed to simply the relationship between the exposure at a single time point and the outcome. The test was whether, on average, the association between alcohol, cigarettes smoking or cannabis use at baseline and later violence varied with age over the entire follow-up period, and to quantify that variation.

POPULATION AVERAGE MODELS

Generalised estimating equation (GEE) modelling was used to fit logistic regression population averaged models. Unlike the logistic regression models presented above which produced an estimate of the simple relationship between alcohol at wave I and violence at a subsequent time point, such that information from subsequent time points was combined into a single composite variable, the GEE approach accommodates multiple observations at different time points. As stated in the methods, the GEE model is a type of population average model, which means that it describes changes in the population mean of an outcome given unit changes in exposure across all people observed, while accounting for non-independence of observations (due to there being multiple observations within individuals). GEE therefore provides an average of the estimates for the association between

exposure at wave outcome and I at each of waves II-IV. By specifying an interaction with time in the model, it is possible to investigate whether the relationship between alcohol and violence varies as a linear function of length of time between exposure and outcome. Put simply, I wanted to find out whether the effect of alcohol at wave I has a weaker, constant or stronger effect on violence emerging at any later wave as the cohort aged. As before, all those who had already been violent at wave I were excluded from the analyses. An exchangeable correlation structure of the residuals was specified with robust standard errors (see methods section for explanation). There was very little difference in the standard errors between models specified, with or without robust standard errors, suggesting that the exchangeable correlation structure was appropriate for these models.

Table 31 shows the crude and adjusted odds ratios between alcohol use (divided into overall quantity of alcohol consumed, frequency of use, number of drinks usually consumed, frequency of binge drinking, or frequency of getting very drunk) and incident violence. With regard to the overall quantity of alcohol consumed, there was a significant linear association with violence (OR 1.35) after adjusting for selected confounders. There was no significant association between the length of time elapsed since wave I and violence (OR 1.00, 95% CI 0.98-1.02), nor was there an interaction between quantity of alcohol consumed and length of time on the odds of violence (OR 0.99, 95% CI 0.98-1.00).

The strongest relationship was between the number of drinks consumed on each occasion and later violence. After controlling for confounders, those that drank between one and 4 drinks each occasion had one and a half time the odds of being violent, and those who drank 5 or more were twice as likely to be violent. The linear relationship was significant, with an odds ratio of 1.35 (95% CI 1.12-1.63).

As before, there was no significant relationship with time, or an interaction between number of drinks consumed and time (odds ratios of 1 in both cases). There was evidence of a relationship between both frequency of binge drinking and violence and frequency of getting very drunk and violence. With regard to binge drinking, the relationship was evident among those who binge drank up to once a month had a higher rate of subsequent violence. Those who binge drank at a higher frequency also showed a trend towards higher risk of violence, but these results were not statistically significant. The overall trend however was marginally significant (OR 1.12, 95% CI 1.0-1.25).

With regard to the frequency of getting very drunk, although in most frequency categories there was no evidence of a significant association, there was weak evidence of an overall trend between frequency of getting very drunk and violence (OR 1.13, 95% CI 1.0-1.27). There was no evidence of an association with time or an interaction effect between exposure and time. This indicates that the risk associated with alcohol consumption at wave I with respect to violence at either wave II, II or IV is constant.

Table 31. Population averaged models of relationship between alcohol use at wave I and onset of violence reported at waves II-IV

Alcohol use	% Incident violence N=9,345	Logistic regression Odds ratio (95% CI) Crude	p	Adjusted*	p
Overall quantity of alcohol consumed					
0 (none)	1.97	1		1	
1	2.70	1.37 (0.96-1.97)		1.64 (1.08-2.50)	
2	2.75	1.40 (0.99-2.00)		1.39 (0.85 -2.32)	
3	4.04	2.09 (1.42-3.06)		2.65 (1.37-4.03)	
4	3.03	1.56 (0.97-2.50)		1.57 (0.79-3.14)	
5 (highest)	3.90	2.02 (1.36-2.99)		1.74 (0.93-3.24)	
Linear trend		1.16 (1.09-1.23)	<0.001	1.35 (1.02-1.27)	0.023
Number of drinks usually consumed each occasion					
0	1.96	1		1	
1-4	2.72	1.39 (1.07-1.81)		1.56 (1.07-2.59)	
5-10	4.10	2.13 (1.55-2.95)		2.21 (1.33-3.65)	
11 or more	3.56	1.85 (1.16-2.93)		2.04 (1.03-4.02)	
Linear trend		1.34 (1.20-1.50)	<0.001	1.35 (1.12-1.63)	<0.001
Frequency of drinking alcohol in last year					
Never	1.97	1		1	
1 or 2 days in past year	3.08	1.58 (1.18-2.12)		1.68 (1.14-2.48)	
Once a month or less	2.94	1.50 (1.04-2.17)		1.83 (1.12-2.97)	
2 or 3 days a month	3.02	1.55 (1.01-2.36)		1.27(0.67-2.39)	
1 or 2 days a week	4.01	2.08 (2.37-3.15)		2.08 (1.06-4.08)	
3 + days per week	3.25	1.68 (0.94-3.01)		0.75 (0.33-1.67)	
Linear Trend		1.16 (1.09-1.24)	<0.001	1.08 (0.96-1.21)	0.196
Frequency of binge drinking in last year					
Never	2.06	1			
1 or 2 days in past year	3.41	1.67 (1.21-2.32)		1.81 (1.15-2.88)	
Once a month or less	4.82	2.40 (2.52 -3.78)		2.04 (1.14-2.66)	
2 or 3 days a month	3.09	1.51 (0.89-2.56)		1.31 (0.63-2.73)	
1 or 2 days a week	2.96	1.45 (0.84-2.49)		1.29 (0.68-2.40)	
3 or more days per week	6.00	3.05 (1.82-5.11)		1.78 (0.78-4.04)	
Linear trend		1.22 (1.14-1.32)	<0.001	1.12 (1.0-1.25)	0.050
Frequency of getting very drunk in last year					
Never	2.10	1		1	
1 or 2 days in past year	3.24	1.53 (1.06-2.22)		2.11 (1.35-3.30)	
Once a month or less	3.42	2.62 (1.08-2.41)		1.70 (0.89-3.20)	
2 or 3 days a month	3.64	1.72 (1.02-2.92		1.64 (0.92-2.91)	
1 or 2 days a week	3.68	1.76 (1.07-2.88)		2.02 (0.94-4.37)	
3 or more days per week	4.18	2.02 (0.99-4.11)		0.98 (0.37-2.58)	
Linear trend		1.19 (1.09-1.28)	<0.001	1.13 (1.0-1.27)	0.051

* Adjusted for the interaction effect between the exposure and elapsed time up to each wave, age at wave I, gender, ethnicity, IQ, depression, impulsivity, temper, cigarette smoking, cannabis use, neighbourhood disadvantage and neighbourhood violent crime wave I. Calculated using wave IV sample weights on subgroup of individuals who were not violent at wave I

IS AMOUNT OF ALCOHOL CONSUMED EACH OCCASION MORE IMPORTANT IN RISK OF VIOLENCE?

As in the previous analyses (results section II), both frequency of drinking and number of drinks usually consumed were entered into the model simultaneously to test their relative importance with respect to incident violence. As in the previous analyses, drinking frequency was not associated with violence, however the number of drinks usually consumed was associated with later violence (OR 1.45, see Table 32).

Table 32. Population average models of the relationship between frequency of alcohol consumption and number of drinks consumed with subsequent onset of violence

	Violence at Waves II-IV Crude		Violence at Waves II-IV Adjusted (n=7641)	
	OR (95% CI)	p	OR (95% CI)	p
Number of drinks usually consumed each occasion (per drink)	1.34 (1.06-1.70)	0.015	1.45 (1.10-1.91)	0.008
Frequency of drinking alcohol in last year	1.04 (0.90-1.21)	0.591	0.93 (0.77-1.11)	0.419

* Adjusted for the interaction effect between the exposure and elapsed time up to each wave, age at wave I, gender, ethnicity, IQ, depression, impulsivity, temper, alcohol use, neighbourhood disadvantage and neighbourhood violent crime at wave I. Calculated using wave IV sample weights on subgroup of individuals who were not violent at wave I

CIGARETTE SMOKING AND CANNABIS USE

The population averaged odds ratios for the effect of cigarette smoking and violence were then calculated in a similar way. In the unadjusted analyses there were 30,560 observations from 7672 participants included in the analysis; in the adjusted analyses, there were 21,958 observations from 5,507 participants included in the analyses. As shown in Table 33, when smoking was divided into 3 categories, the rate of later onset of violence was higher among those who were already smoking 1-60 cigarettes per month by the time of the wave I interviews than among the non-smokers (OR 1.51). Those who smoked more heavily had an odd of violence roughly 1.6 times higher than the non-smokers (range between one and two and a half. The linear trend for number of cigarettes smoked, however,

was not significant. This indicates that being a smoker is associated with violence, irrespective of the number of cigarettes smoked. There was no significant interaction between smoking and time in years, however there was a significant association between time and violence in this model (OR 0.99 95% CI 0.99-0.99, p=0.037) indicating that the odds of violence decreases by 1% for every year that elapsed.

With regard to smoking cannabis, there was no increased risk of subsequent onset of violence, regardless of categorised quantity, compared to those who did not use it at all. Neither, therefore, was there an association by overall linear trend.

Table 33. Population average models of relationship between cigarette smoking and cannabis use at wave I and onset of violence reported at wave II-IV

	Number and weighted Proportion of subjects violent after Wave I	Logistic regression Odds ratio (95% CI)			
		Crude	p	Adjusted*	p
Total Number of times smoked cigarettes in last 30 days					
0	692 (2.1%)	1 (reference)		1 (reference)	
1-60	141 (3.6%)	1.75 (1.27-2.41)		1.51 (1.02-2.21)	
61 or more	142 (3.9%)	1.90 (1.42-2.56)		1.60 (0.99-2.57)	
Linear trend (per 10 cigarette)		1.01 (1.002-1.02)	0.005	1.004 (0.99-1.02)	0.432
Number of times smoked cannabis in last 30 days					
1	771 (7.5%)	1 (reference)			
1-10	146 (14.6%)	2.27 (1.62-3.18)		1.22 (0.80-1.87)	
11 or more	45 (8.8%)	1.32 (0.72-2.43)		0.59 (0.68-1.07)	
Linear trend (per 10 times)		1.01 (0.97-1.03)	0.716	0.85	0.176

* Age Gender, Ethnicity, IQ, Delinquency, Depression, Temper, Family Conflict, Family Structure, peer substance use, Neighbourhood Violent Crime, Neighbourhood Disadvantage and population density, and alcohol use. Calculated on subgroup of individuals who were not violent at Wave I using wave IV sample weights

CHAPTER 14 LONGITUDINAL MODELS OF RELATIONSHIP BETWEEN ALCOHOL, CIGARETTE SMOKING OR CANNABIS USE AND VIOLENCE IN ENTIRE COHORT

In contrast to the methods used previously, which considered average effects within the cohort with respect to incident violence, this section now considers the behaviour of all individuals within the sample, and investigates differences between individuals over time. Whereas before, the average association between exposure and outcome at each wave was modelled, in this section, the individual level of exposure (levels of alcohol consumption, cigarette smoking or cannabis use) over each wave were modelled along with change in reported violence. This approach therefore accommodates the change in exposure over time within individuals not simply the average exposure within the sample., as well as the change in outcome over time.

As this section investigates relationships in the whole cohort whereas previous sections concentrated on the cohort who were not violent before wave I, I begin with a comparison of these two groups. I then begin to explore trajectories of violence within the entire cohort as a prelude to construction of appropriate longitudinal models. I then explore the most appropriate longitudinal model for the data by comparing preliminary analyses using fixed and random effects. I then proceed with random-effects models modelling the effects of alcohol, smoking and cannabis use on violence in the same model. I then plot the marginal effects of each of these exposures on violence. Finally, I investigate these relationships on the secondary measures of violence for comparison.

COMPARISON OF COVARIATES BETWEEN THOSE WHO REPORTED VIOLENCE BEFORE WAVE I AND THOSE WHO DID NOT

The prevalence of covariates and outcomes were compared in the two cohorts. There were significant differences between those who were violent before wave I and those that were not. Over two thirds of the cohort of individuals who were violent before wave I were males (69.9%), whereas less than half (44.8%) of the group who were not violent before wave I were males. The violent group were slightly but statistically significantly younger at wave I (16.07 compared with

16.18). There were differences too in the ethnic composition of the groups. The violent group was comprised of 57% white, 28% black, 5.2% Asian/pacific and 9% other ethnicities. By contrast, the non-violent group had proportionally more White (63%), fewer Black (21 %), more Asian/Pacific (7%) and fewer other ethnicities (7.5%). The violent group were more delinquent (median delinquency score 5 compared with 2), had friends who used more substances, had a higher depression score (12 compared with 10) had a lower mean IQ (98 compared with 100.1), were more impulsive (mean 2.17 compared with 2.24) and came from a neighbourhood with a higher violent crime rate (see Table 34).

Table 34. Comparison of covariates among those who reported violence before wave I and those who reported no violence before wave I

	Violent before wave I			
	No (n=16,761) Mean / median / proportion	Yes (n=3,871) Mean / median / proportion	Test statistic	P
Male[a]	44.8	69.9	796.9	<0.001
Age[b]	16.18	16.07	3.63	<0.001
Delinquency score[c]	2	5	-43.3	<0.001
IQ[b]	100.1	98.0	8.17	<0.001
Depression[c]	10	12	-15.9	<0.001
Neighbourhood disadvantage[b]	-0.02	0.10	-7.07	<0.001
Neighbourhood violent crime[b]	8.78	9.33	-4.49	<0.001
Impulsivity[b]	2.17	2.24	-6.09	<0.001
Temper[a]	28.5	43.3	272.0	<0.001
Peer substance use at wave I[c]	1	3	-27.9	<0.001
Peer substance use at wave II[c]	2	4	-20.3	<0.001

[a]Proportion and chi squared statistic [b]Mean and t-test statistic [c]Median and Mann Witney U statistic

COMPARISON OF PREVALENCE OF ALCOHOL CONSUMPTION, SMOKING AND CANNABIS USE BETWEEN THOSE WHO REPORTED VIOLENCE BEFORE WAVE I AND THOSE WHO DID NOT

There were significant differences between groups in the prevalence of alcohol use, cigarette smoking and cannabis use (See Table 35). The proportion of people who were violent at each wave is shown in Figure 9 and Figure 10. Figure 9 show the proportion of people who were violent among those violent before wave I, and shows a sharp decline in violence between wave I and II, followed by a gradual,

almost linear decline from wave II to wave IV. Figure 10 shows that among those who were not violent before wave I, a small proportion become violent before wave II. A similar proportion is violent at wave III, and there is a decline by wave IV.

At every wave, among those who reported violence prior to wave I. there was a higher proportion who drank more, smoked or used cannabis compared with those who did not report violence prior to wave I (see Figure 11 and Figure 12 (alcohol), Figure 13 and Figure 14 (cigarette smoking) Figure 15 and Figure 16 (cannabis use), and Table 35).

Figure 9. Proportion who reported violence at each wave among those who were violent before wave I

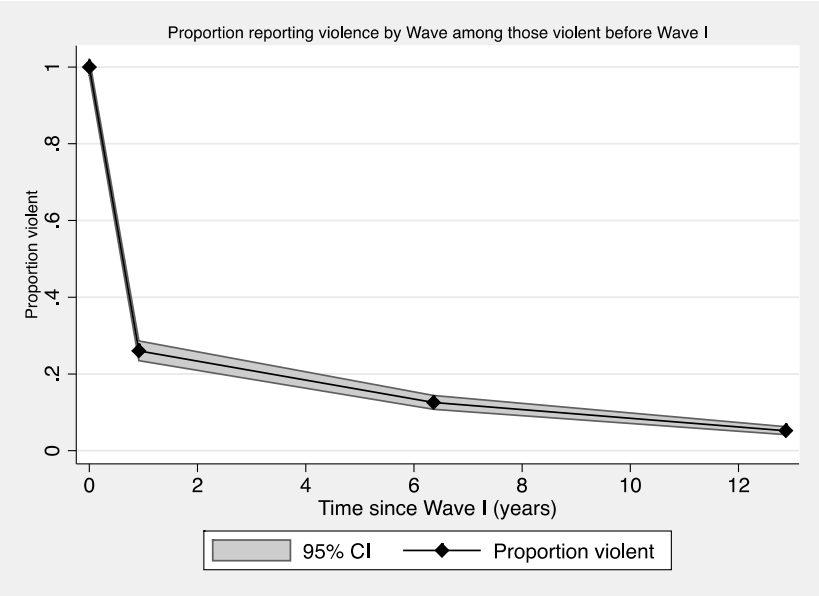


Figure 10. Proportion of people who reported violence at each wave among those who were not violent before wave I

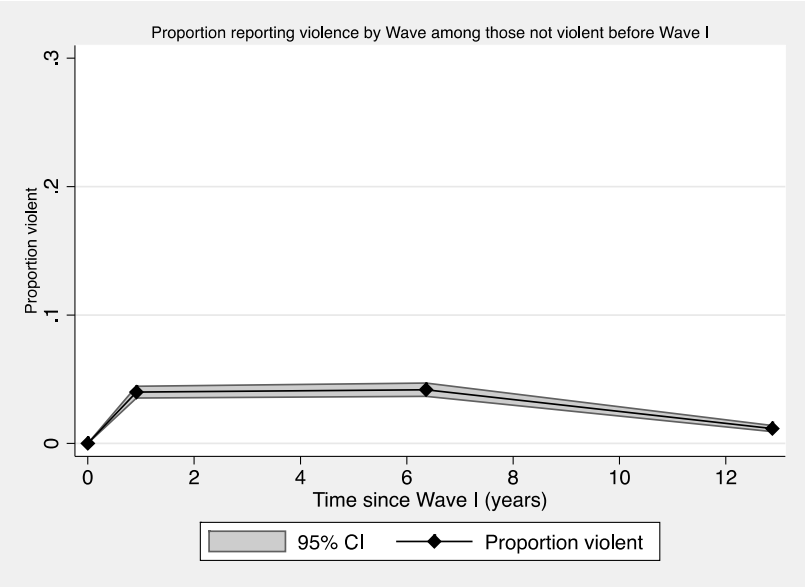


Table 35. Comparison of outcomes among those who reported violence before wave I and those who reported no violence before wave I

	Not violent before wave I		Violent before wave I			
	No.	Weighted %	No.	Weighted %	X^2	p
Wave I						
Number of drinks						
None	9,345	56.1	1,599	40.0		
1-4	4,725	28.0	1,099	28.2		
5 or more	2,488	15.9	1,130	31.8		
					507	<0.001
Frequency of drinking						
None	9,307	55.6	1,579	38.9		
Less than once a week	6,031	36.4	1,570	41.1		
Once a week or more	1,304	8.1	715	19.8	558.0	<0.001
Smoking	3,829	24.9	1,482	41.1	177.3	<0.001
Cannabis use	1,902	11.5	1,019	27.2	29.31	<0.001
Wave II						
Number of drinks						
None	9,345	58.0	1,599	45.1		
1-4	4,725	23.8	1,099	23.6		
5 or more	2,488	18.2	1,130	31.2	67.7	<0.001
Frequency of drinking						
None	6,989	57.5	1,273	44.0		
Less than once a week	3,879	33.3	961	36.4		
Once a week or more	1,067	9.2	511	19.5	63.0	<0.001
Smoking	3,486	32.1	1,150	45.1	83.8	<0.001
Cannabis use	1,583	13.7	717	28.5	147.0	<0.001
Wave III						
Number of drinks						
None	9,345	27.0	1,599	27.1		
1-4	4,725	43.9	1,099	36.2		
5 or more	2,488	29.1	1,130	36.6		
					12.6	<0.001
Frequency of drinking						
None	3,403	57.9	727	42.0		
Less than once a week	5,576	33.4	1074	37.4		
Once a week or more	3,280	8.8	824	20.6		
					70.1	<0.001
Smoking	3,644	33.0	1,088	45.1	70.0	<0.001
Cannabis use	2,431	22.5	765	32.6	50.3	<0.001
Wave IV						
Number of drinks						
None	3,626	26.2	801	26.7		
1-4	7,051	54.4	1,267	42.6		
5 or more	2,094	19.2	723	30.7		
					31.8	<0.001
Frequency of drinking						
None	3,626	26.1	801	26.2		
Less than once a week	5,493	43.2	1,068	42.1		
Once a week or more	3,740	30.8	946	31.2		
					5.8	0.004
Smoking	4,207	35.8	1,348	51.9	123.1	<0.001
Cannabis use	1,812	16.1	691	25.8	42.1	<0.001

Figure 11. Overall quantity of alcohol consumed among those who were violent before wave I

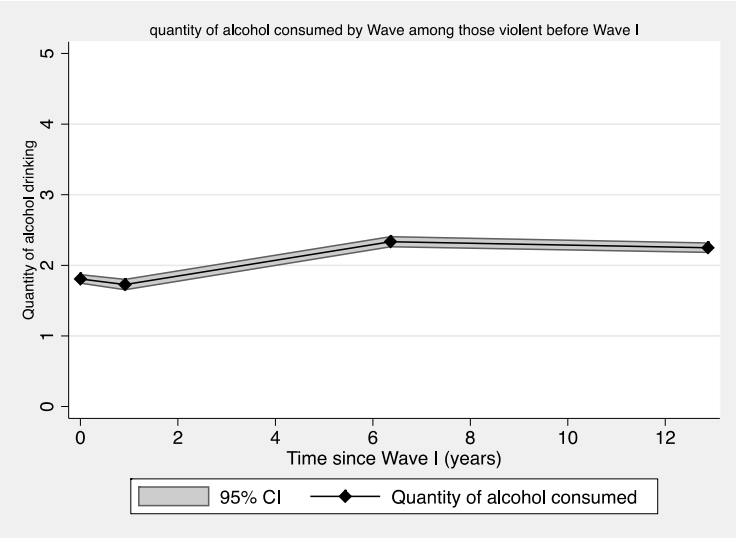


Figure 12. Overall quantity of alcohol consumed among those who were not violent before wave I

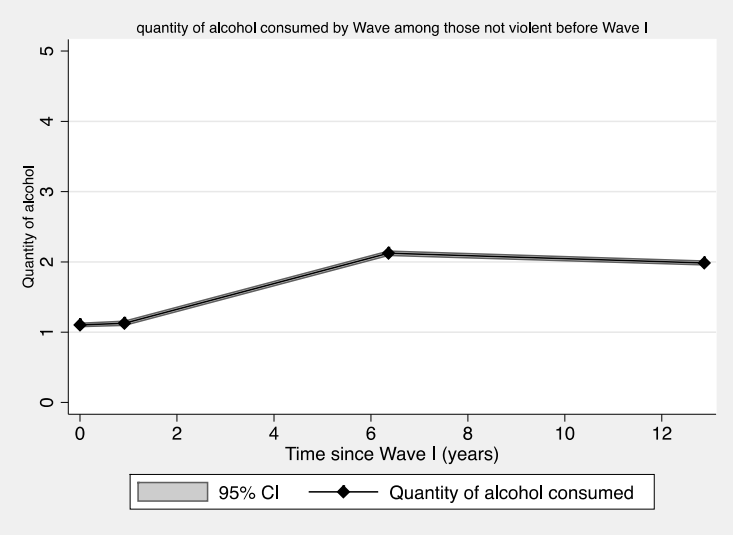


Figure 13. Mean and 95% CI of number of cigarettes smoked per month among those who were violent before wave I

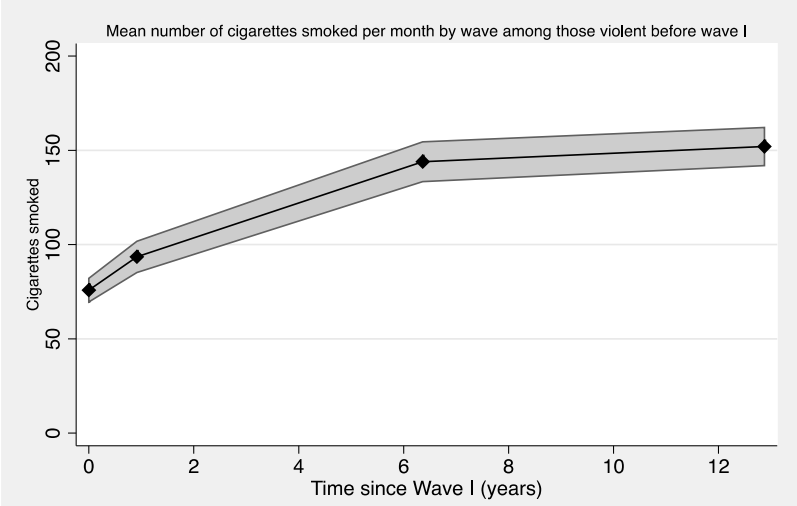


Figure 14. Mean and 95% CI of number of cigarettes smoked per month among those who were not violent before wave I

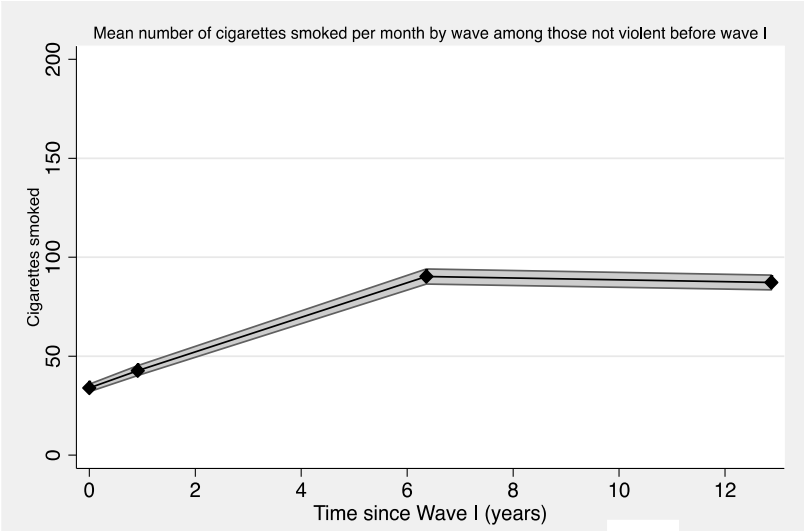


Figure 15. Proportion of people who used cannabis among those who were violent before wave I

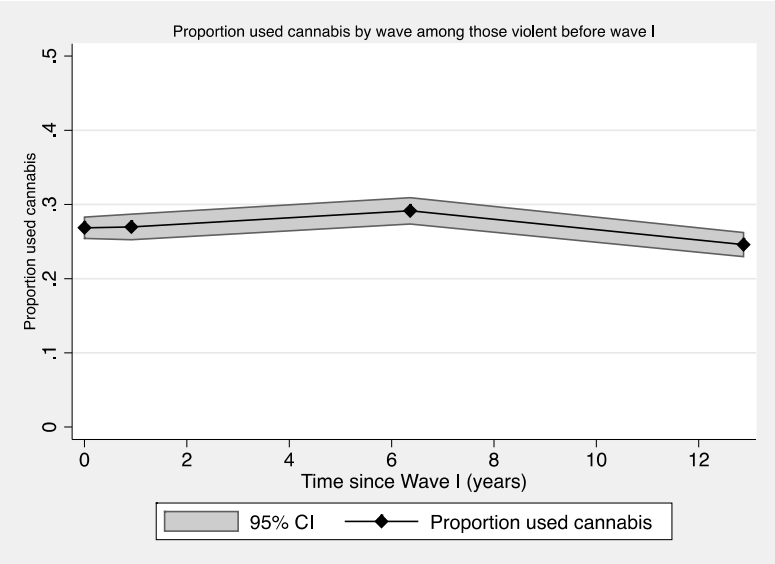
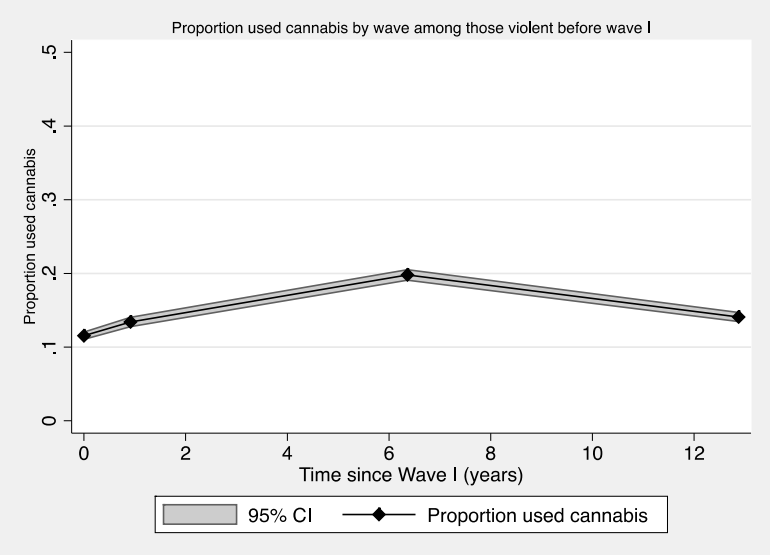


Figure 16. Proportion of people who used cannabis among those who were not violent before wave I



In order to begin to construct a model that describes the trajectory of violence, a random sample of individual trajectories was first examined by plotting rates of violence by wave for a sample of 100 randomly selected participants. Because a large proportion of participants were not violent at any wave, and therefore have flat trajectories, participants were sampled from among those who were violent on at least one time point. Their frequency of violence at each wave was plotted and the results of all 100 were combined in a single panel in a trellis plot, for ease of viewing. As can be seen in Figure 17, although there is some variation, the tendency is towards a lower frequency of violence over time within individuals.

Next, the trajectories were explored to test for linearity, by plotting a fitted (regression) line through the observed points. The panel of 100 randomly selected individuals with fitted values is shown in Figure 18. This shows that the trajectories are approximately linear, and with a downward slope. It can be seen from the fitted trajectories that there is variation in the starting point (intercept), but less variation in the rate of change (slopes).

Figure 17. Trellis plot of trajectories of violence for random sample of 100 participants who had reported having been violent on at least one wave

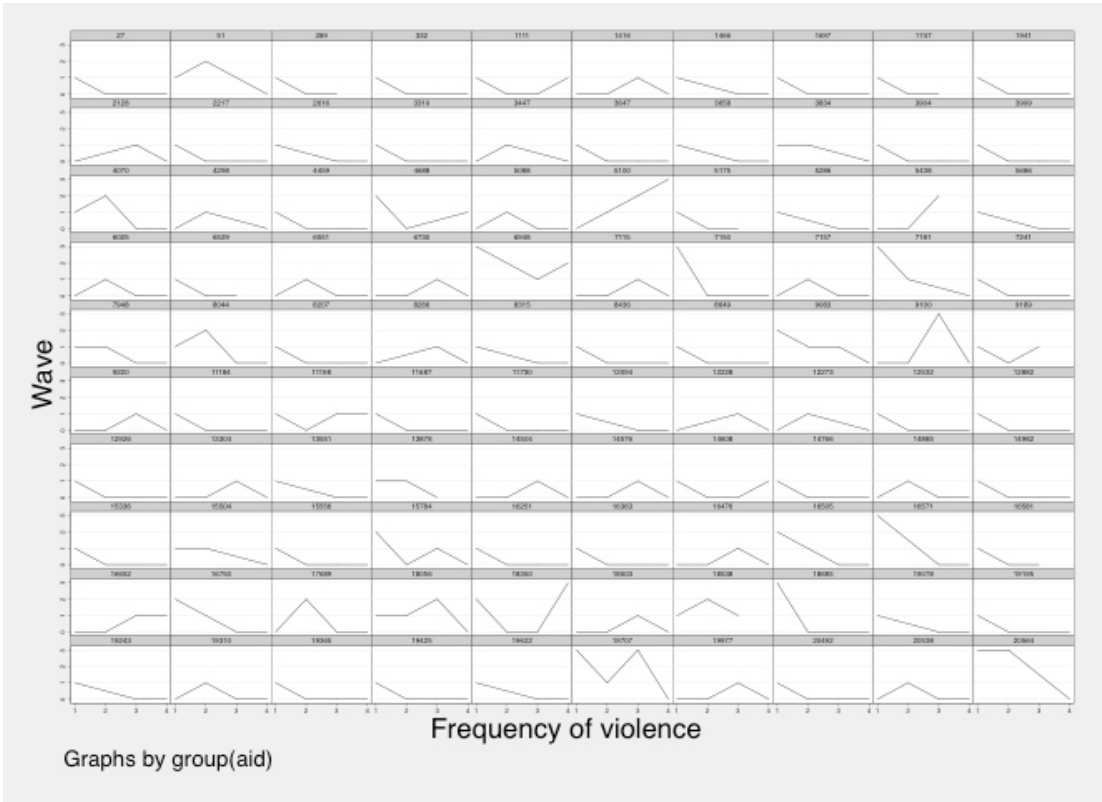
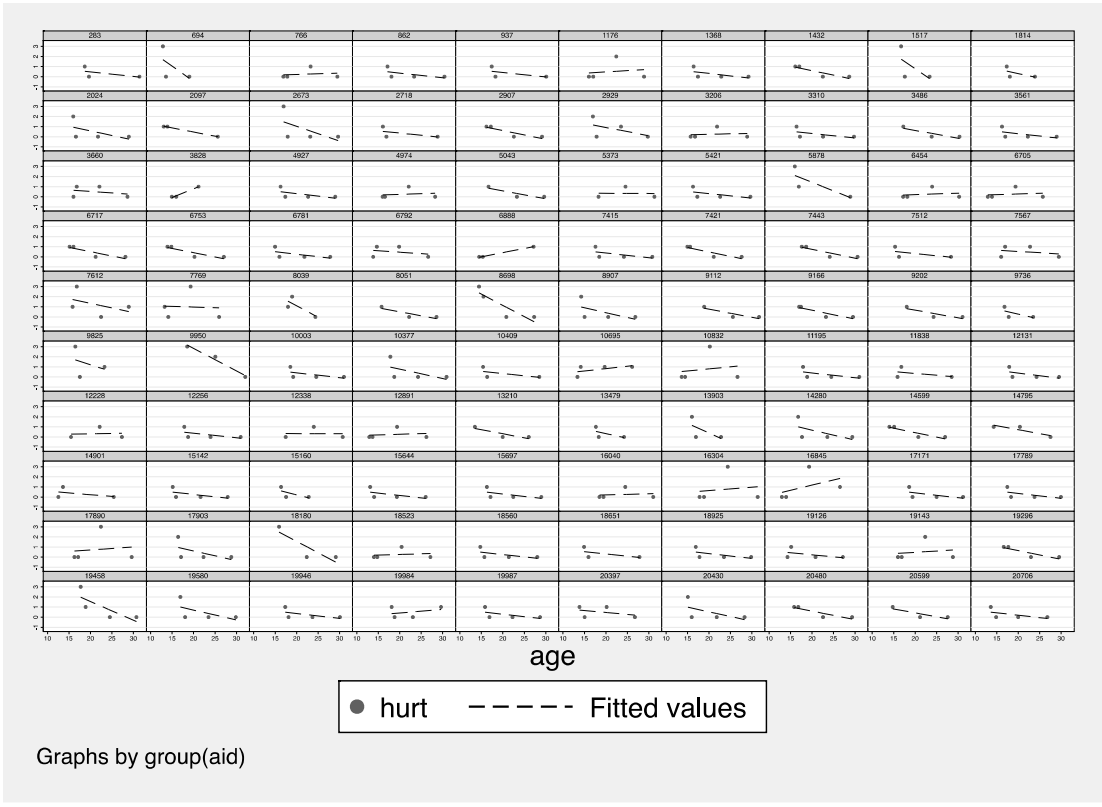


Figure 18. Fitted linear trajectories of violence for random sample of 100 participants with onset of violence reported on at least one of waves II-IV



CHOICE OF VARIABLES FOR ESTIMATING THE LONGITUDINAL RELATIONSHIP BETWEEN ALCOHOL AND VIOLENCE

For the purpose of exploring change in the relationship between alcohol and violence, the number of drinks usually consumed on each occasion was used as in the previous section it was shown to be an important factor exposure variable for alcohol. Just three categories were used, for ease of interpretation. They were: no alcohol, 1-4 drinks, and 5 or more drinks each occasion. As in previous sections, the outcome measure was - physical harm to others needing treatment.

SELECTION OF THE MOST APPROPRIATE MODEL

In determining whether a fixed- or random-effects model would be more appropriate, exploratory analyses were carried out on the entire cohort. Here, a logistic fixed-effects model with interaction between number of drinks consumed and age was fitted first on the entire cohort. Age was centred at the mean age at wave1 (age 16). This means that the odds ratios produced in the models represent the effect when the individual was 16. Age squared was also included in the model to allow for the non-linear effect of age. A likelihood ratio test confirmed that there was a significant improvement in model fit when age squared was included, and therefore it was retained in the model. The results of this model are shown in Table 36.

Table 36. Fixed-effects model showing relationship between number of drinks usually consumed and violence, with drinking or violence starting in any wave (n=3,378)

Violence	OR	95% CI	p
Number of drinks usually consumed			
0	(Reference)		
1-4	1.37	1.18-1.79	<0.001
5 or more	1.99	1.68-2.35	<0.001
Age (centered at 16)	0.72	0.67-0.78	<0.001
Age squared	1.00	1.00-1.00	0.01
Number of drinks X age			
0	(Reference)		
1-4	0.98	0.95-1.01	0.147
5 or more	1.01	0.98-1.04	0.232

In this fixed-effects model, only those individuals whose violence changes over time contributed statistically to the analysis. Thus all individuals whose violence was constant were necessarily eliminated. The model in Table 36 therefore represents analysis of only a sub-sample (n=3,378) within the cohort; only around a fifth of those who had data at more than one time-point were included. This increases the risk of exclusion bias, that is that the sample on which the analyses are carried out may be systematically different to the population of interest.

A preliminary random-effects model was then fitted on the same (n=3,378) dataset as just used for the fixed-effects model, for comparison, which also included other factors, which for the purpose of the model were considered time-invariant. Some of the variables are truly time-invariant, such as gender and ethnicity, but others were treated as time invariant even though they were probably not, as only the values given at wave I were used. They were delinquency, depression, IQ, temper, smoking, use of cannabis, use of other drugs, neighbourhood disadvantage, neighbourhood violent crime rate, and neighbourhood population density. This model produced similar results to the fixed-effects model (see Table 37). The results of analyses carried out using both the fixed effects model and the random effects model, showed that those who reported drinking 1-4 drinks each occasion, the odds of violence was 37% higher compared with those who did not drink. The standard error was smaller, and consequently the confidence intervals narrower in the random effects model. The odds of violence was twice as high for those who usually drank 5 or more drinks each occasion compared with those who did not drink (found using both models, but the random-effects model produced a smaller standard error).

My preliminary analyses therefore revealed little difference between the results obtained from either the fixed effects or random effects models, and it was decided to proceed using random-effects models as a more precise model, and to retain a larger sample in the analysis. For these analyses, the entire sample (n=20,748) were eligible for inclusion, even if they provided data at only one time-point as their information could still contribute towards the analyses.

Table 37. Random-effects model showing relationship between number of drinks usually consumed and violence over time regardless of when drinking or violence started (n=3,273)

Violence	OR	95% CI	p
Number of drinks usually consumed			
0	(Reference)		
1-4	1.37	1.21-1.55	<0.001
5 or more	1.96	1.71-2.25	<0.001
Age (centered at 16)	0.85	0.79-0.92	<0.001
Age squared	1.00	1.00-1.00	0.176
Number of drinks X age			
0	(Reference)		
1-4	0.96	0.94-0.99	0.005
5 or more	1.00	0.97-1.02	0.835

RANDOM-EFFECTS MODELS

ALCOHOL

The random-effects model which modelled the effect of individual change in levels of exposure on the outcome while adjusting simultaneously for time-variant and time–invariant confounders (see Table 38) showed that, overall, drinking 1-4 drinks on each occasion was associated with a 40% increase in risk of violence (OR 1.4, 95% CI 1.2-1.6). Heavier drinking, (more than 5 drinks on each occasion) was associated with over twice the odds of violence compared with non-drinkers (OR 2.1, 95% CI 1.8-2.5).

Figure 19 shows the predictive marginal effect of alcohol (plotted by categories of no alcohol, 1-4 drinks and 5 or more) on the probability of violence from age 12-30. As can be seen from

Figure 19, drinking 1-4 drinks or 5 or more drinks on each occasion is associated with a significantly higher probability of violence between age 12 and 18. Between age 18 and 30, there is no additional risk of violence among those who drank 1-4 drinks each occasion compared with those who drank no alcohol. Those who usually drank 5 or more drinks had a significantly higher probability of violence than non- drinkers throughout the period of observation, but the trajectories tend to converge with increasing age. The effect of alcohol on the probability of violence therefore is most potent the younger the individual.

Figure 19. Predictive marginal effects (with 95% CIs) of violence for number of drinks usually consumed, age 12-30

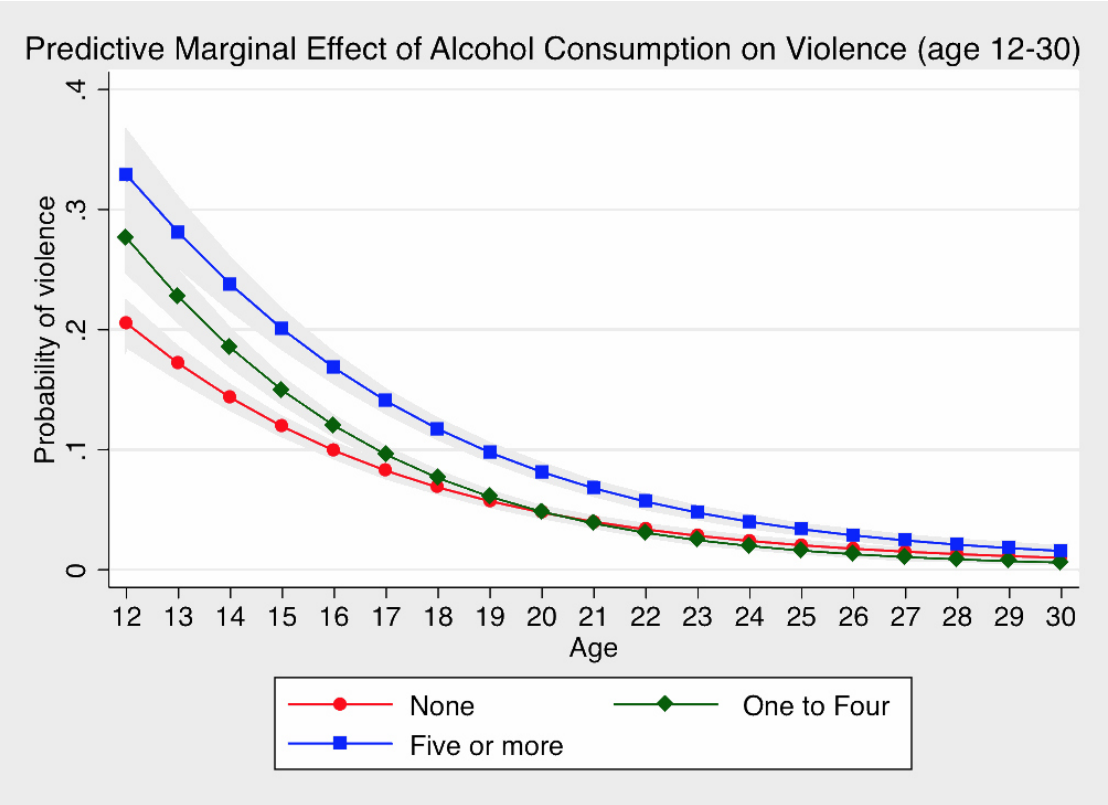
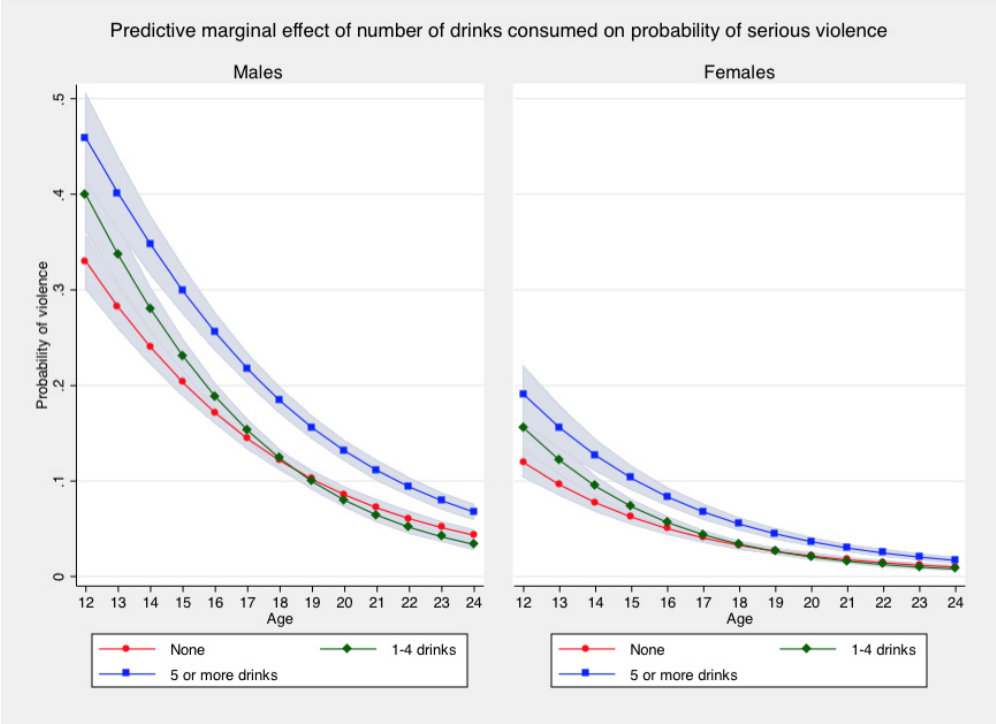


Table 38. Random-effects model showing the estimated effects of quantity of alcohol, cigarette smoking and cannabis use (time variant) on violence, adjusted for time-invariant covariates including interaction terms (n=15,057)

Violence	OR	95% CI	p
Number of drinks usually consumed			
0	(Reference)		
1-4	1.36	1.18-1.58	<0.001
5 or more	2.14	1.80-2.54	<0.001
Number of times smoked cigarettes in last month			
None	1 (reference)		
1-60	1.00	0.84-1.20	0.94
61 or more	1.52	1.27-1.82	<0.001
Number of times used cannabis in last 30 days			
None	1 (Reference)		
1-10	1.42	1.20-1.68	<0.001
11 or more	1.74	1.40-2.15	<0.001
Age (centered at 16)	0.78	0.76-0.80	<0.001
Age squared	1.00	1.00-1.00	0.137
Number of drinks X age			
0	(Reference)		
1-4	0.95	0.93-0.97	<0.001
5 or more	0.99	0.96-1.02	0.392
Ethnicity			
White	(Reference)		
Black	1.70	1.51-1.92	<0.001
Asian	0.68	0.55-0.85	<0.001
Other	1.20	1.02-1.41	0.029
Gender (female)	0.24	0.22-0.27	<0.001
Delinquency	1.17	1.15-1.19	<0.001
IQ	0.99	0.98-0.99	<0.001
Temper	1.55	1.41-1.69	<0.001
Depression	1.02	1.01-1.02	<0.001
Parents argue	0.93	0.88-0.98	0.007
Peer substance use	1.09	1.06-1.11	0.009
Neighbourhood violent crime rate	1.02	1.01-1.02	<0.001
Neighbourhood disadvantage	1.09	1.04-1.14	<0.001
Alcohol X delinquency			
0	1		
1-4	0.97	0.95-0.99	0.007
5 or more	0.97	0.94-0.99	0.001
Cigarettes X delinquency			
None	1		
1-60	1.02	1.0-1.04	0.112
61 or more	0.97	0.95-0.99	0.005
Cannabis X delinquency			
None	1		
1-60	0.98	0.96-0.98	0.026
61 or more	0.98	0.96-1.01	0.212

The predictive marginal effects were then plotted by gender. As can be seen in Figure 20, the probability of violence is greater in males than in females, however the pattern is similar in both genders; drinking 5 or more drinks each occasion is associated with a higher probability of violence from age 12 continuing into mid 20s and beyond, whereas consuming 1-4 drinks is not associated with a significantly higher probability of violence compared with non-drinkers.

Figure 20. Predictive marginal effects (with 95% CIs) of violence for number of drinks usually consumed each occasion, age 12-24, by gender

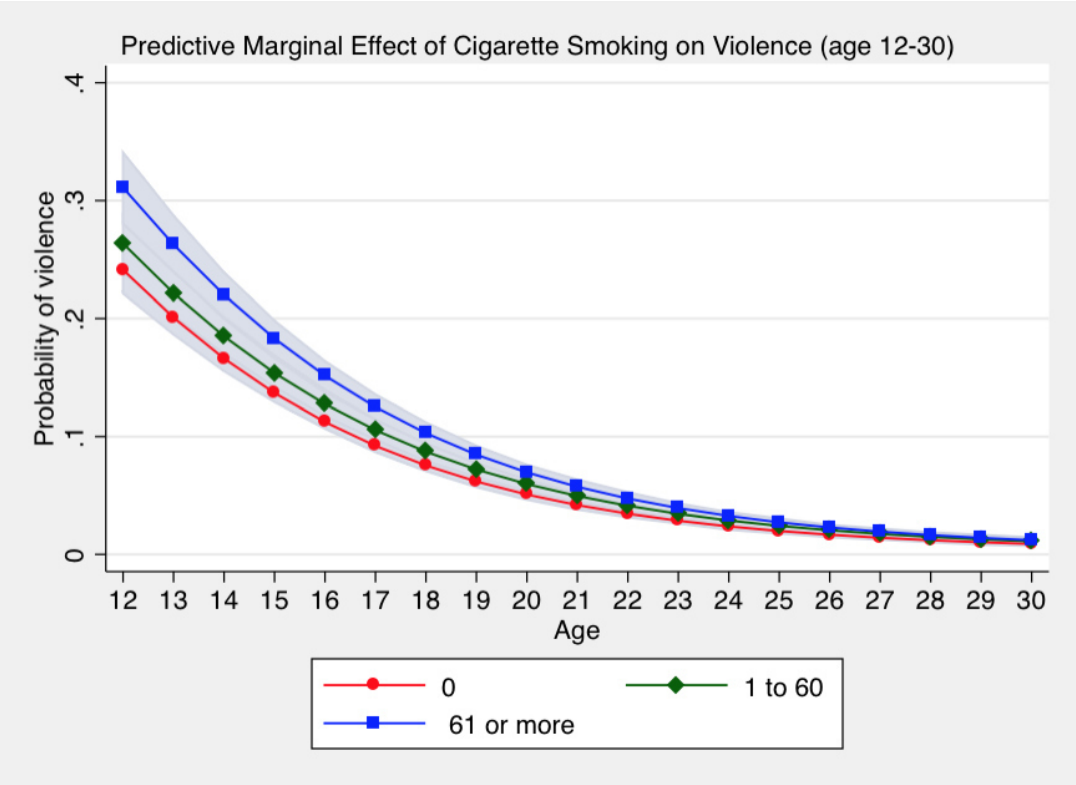


CIGARETTE SMOKING AND VIOLENCE

The random effect model (Table 38) showed that a given individual who smoked 61 or more cigarettes in a month had an odds of violence 1.7 times greater than a similar non-smoker (OR 1.8, 95% CI 1.5-2.1). Those who smoked 1-60 cigarettes had no higher risk of violence than non-smokers.

Using data from the model in Table 38, the predictive marginal effect for cigarette smoking was plotted over the ages 12-30 and presented in Figure 21.

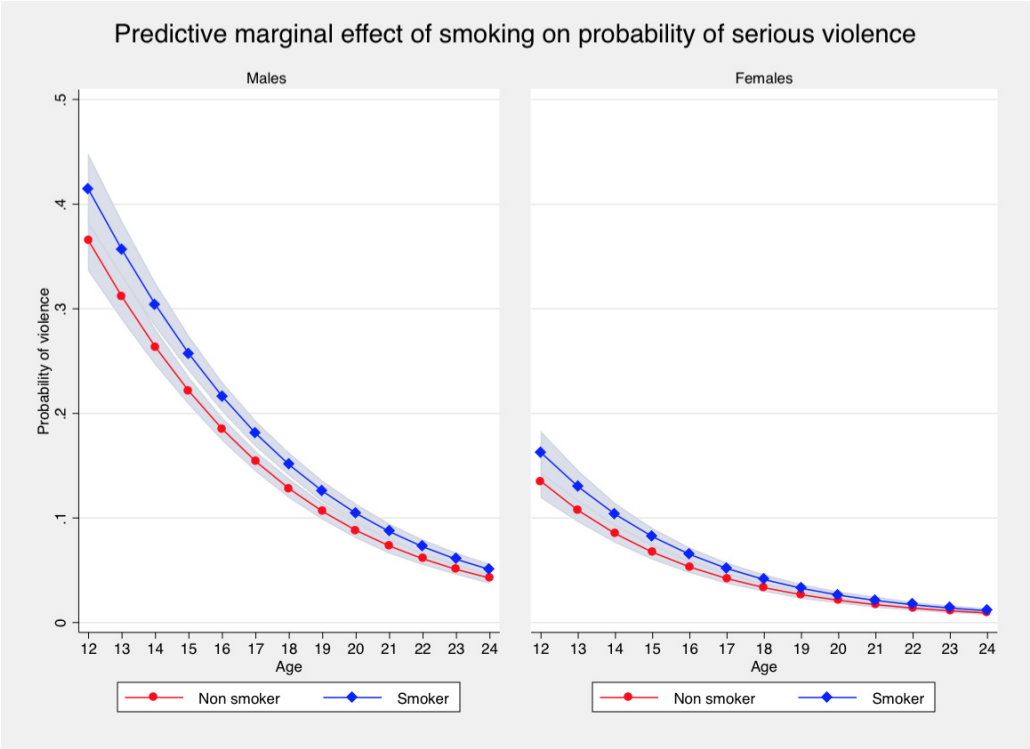
Figure 21. Predictive marginal effects of violence for number of cigarettes smoked in a month



The predictive marginal effect of smoking shows that the greatest effect is at the younger age. At age 12 the probability of violence increases from approximately 0.22 to 0.3; the trajectories tend to converge as participants get older.

The marginal effects were then plotted for males and females separately (see Figure 22). Among males, smokers had a significantly higher probability of violence than non-smokers between age 14 and 19, although the difference was small (approximately 0.01). There was no evidence of difference between smokers and non-smokers among the females.

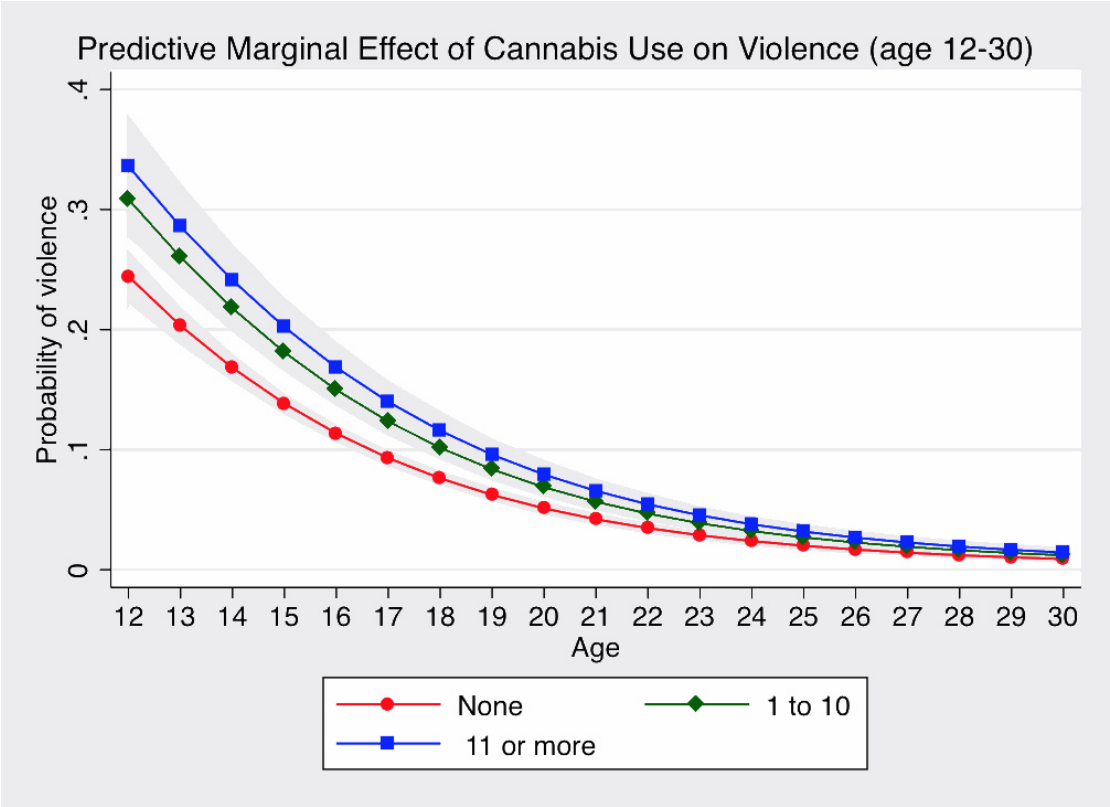
Figure 22. Predictive marginal effects of smoking on violence, by gender



CANNABIS USE AND VIOLENCE

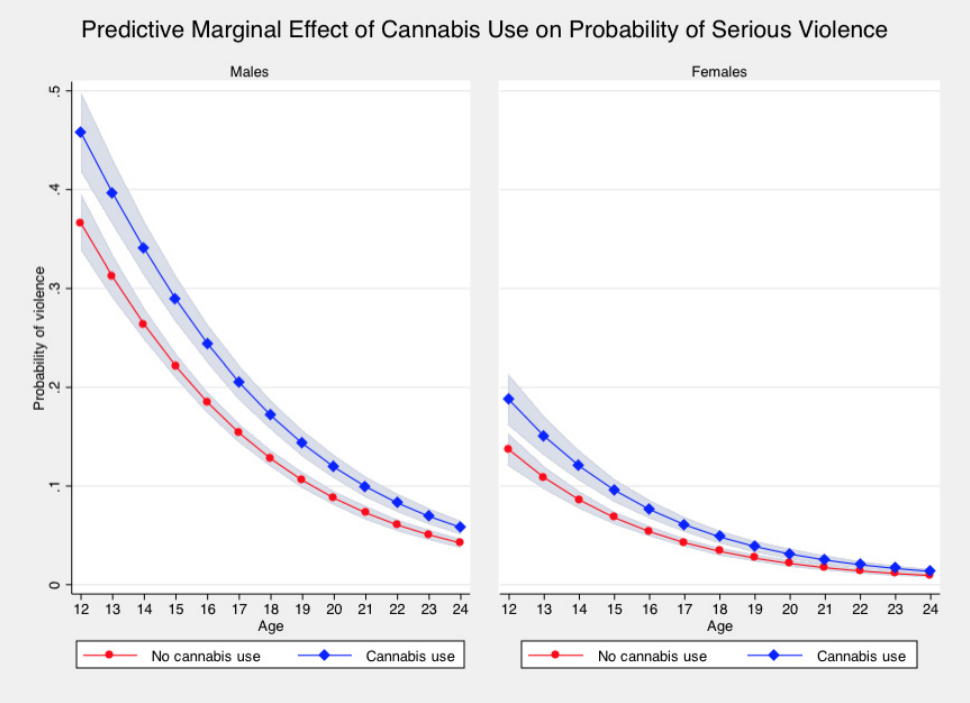
The random-effects model (Table 38) showed that cannabis use was associated with violence. Using cannabis 1-10 times a month was associated with an odds of violence 1.6 times higher than those who did not use cannabis, (OR1.6, 95% CI 1.3-1.9), and those that used it 11 or more times had an odds of violence 1.9 times greater (OR 1.9, 95% CI 1.4-2.5). The predictive marginal effect showed that the categories of using cannabis 1-10 and 11 or more times were very similar in terms of the predictive marginal effects, and both of these were significantly higher probability of violence than the non cannabis users from age 12-22, thereafter the trajectories tended to converge (see Figure 23).

Figure 23. Predictive marginal effects of violence for number of times used cannabis in a month



The marginal effect was then plotted by gender, and showed a similar effect in both males and females, that individuals who used cannabis had a higher probability of violence after controlling for important confounders. The effect was greatest in younger individuals, and become smaller as individuals got older (see Figure 24).

Figure 24. Predictive marginal effect of cannabis use on violence, by gender



SECONDARY MEASURES OF VIOLENCE

The relationship between alcohol and two secondary measures of violence, involvement in a "serious physical fight", and involvement in fighting in a group was then investigated using random-effects modelling as before.

SERIOUS FIGHTING

As shown in Table 39, drinking between 1-4 drinks each occasion was associated with odds of being involved in at least one serious physical fight 1.2 times higher than those who did not drink (95% confidence interval 1.08-1.41). Those who usually drank 5 or more drinks were 1.75 times more likely to be involved in a serious physical fight (95% confidence interval 1.49-2.05).

The predictive marginal effects are shown in

Figure 25. Those who usually drink 5 or more drinks have an increased probability of violence relative to those who do not drink throughout the period age 12-30. Those who drink more moderately have no greater probability of violence than those who do not drink, except in early adolescence when there is a small increase in risk. By age 19 the trajectories of those who drink moderately and those who do not drink converge.

As shown in Figure 26, the effects are similar in both genders, except that the effect of drinking 5 or more drinks each occasion appears to have a greater effect on increasing the probability of violence in males than in females.

Figure 25. Predictive marginal effects of number of drinks consumed on probability of serious fighting

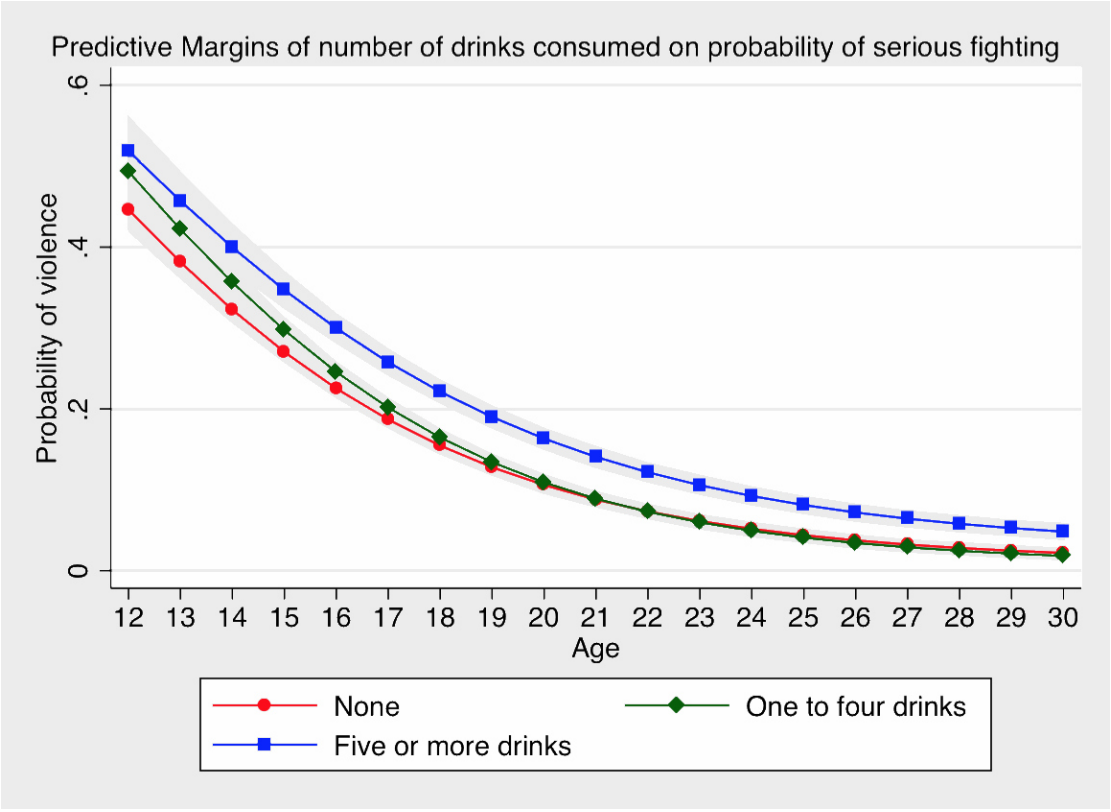
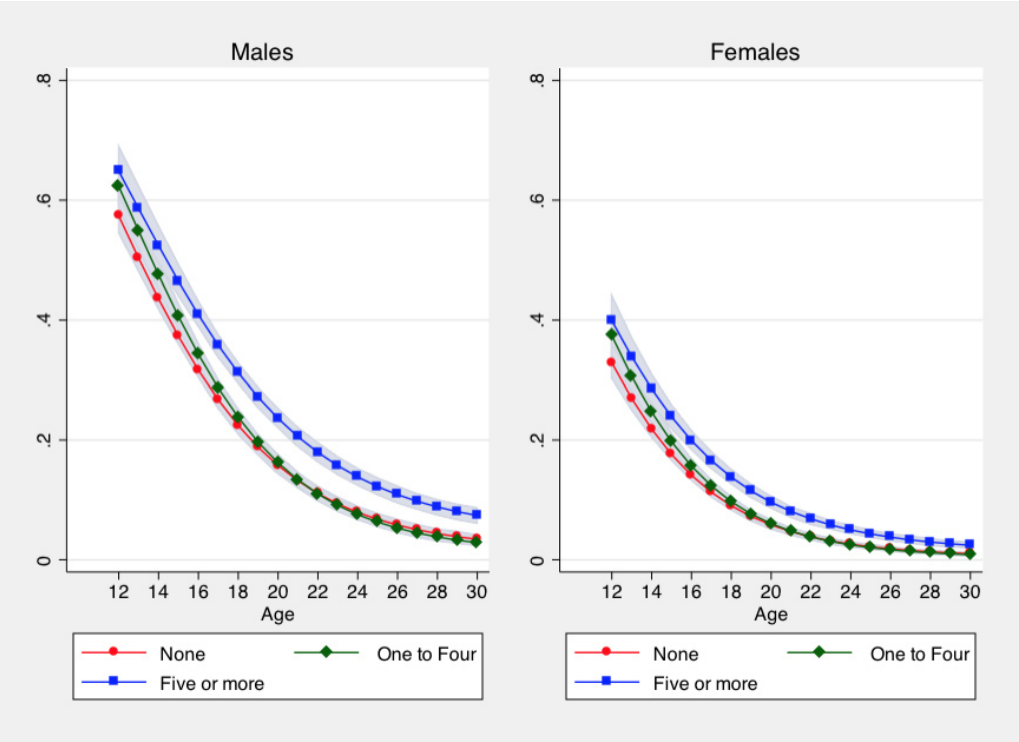


Table 39. Random-effects model showing the estimated effects of quantity of alcohol, cigarette smoking and cannabis use (time variant) on serious fighting, adjusted for time-invariant covariates including interaction terms (n=15,046)

Violence	OR	95% CI	p
Number of drinks usually consumed			
0	(Reference)		
1-4	1.23	1.08-1.41	0.001
5 or more	1.75	1.49-2.05	<0.001
Number of times smoked cigarettes in last month			
None	1 (reference)		
1-60	1.15	0.98-1.34	0.090
61 or more	1.82	1.53-2.16	<0.001
Number of times used cannabis in last 30 days			
None	1 (Reference)		
1-10	1.40	1.19-1.64	<0.001
11 or more	1.30	1.02-1.64	0.030
Age (centered at 16)	0.74	0.71-0.76	<0.001
Age squared	1.00	1.00-1.00	0.137
Number of drinks X age			
0	(Reference)		
1-4	0.98	0.96-1.00	0.064
5 or more	1.03	1.00-1.05	0.008
Ethnicity			
White	(Reference)		
Black	1.79	1.60-2.03	<0.001
Asian	0.78	0.64-0.94	<0.001
Other	1.33	1.14-1.55	0.029
Gender (female)	0.30	0.28-0.32	<0.001
Delinquency	1.17	1.15-1.19	<0.001
IQ	0.99	0.98-0.99	<0.001
Temper	1.72	1.58-1.87	<0.001
Depression	1.02	1.01-1.02	<0.001
Parents argue	0.97	0.92-1.01	0.200
Peer substance use	1.09	1.06	1.11
Neighbourhood violent crime rate	1.02	1.01-1.02	<0.001
Neighbourhood disadvantage	1.14	1.09-1.02	<0.001
Alcohol X delinquency			
0	1		
1-4	0.98	0.96-1.00	0.148
5 or more	0.99	0.96-1.08	0.216
Cigarettes X delinquency			
None	1		
1-60	0.98	0.96-1.01	0.263
61 or more	0.95	0.93-0.97	<0.001
Cannabis X delinquency			
None	1		
1-60	0.98	0.96-0.98	0.026
61 or more	0.98	0.96-1.01	0.212

Figure 26. Predictive marginal effect of number of drinks usually consumed on probability of serious fighting, by gender



GROUP FIGHTING

The association between alcohol consumption and fighting in a group was next analysed. Table 40 shows the results of results of the random-effects model for the number of drinks consumed, and fighting in a group. Those who drank 1-4 drinks each occasion were 1.76 times more likely to engage in a group fight than those who did not drink alcohol (95% confidence intervals 1.54-2.01). Those who drank 5 or more drinks each occasion had an odds of violence 2.58 times higher than the non-drinkers (2.20-3.02).

Table 40. Random-effects model showing the estimated effects of quantity of alcohol, cigarette smoking and cannabis use (time variant) on group fighting, adjusted for time-invariant covariates including interaction terms (n=15,056)

Violence	OR	95% CI	p
Number of drinks usually consumed			
0	(Reference)		
1-4	1.76	1.54-2.01	<0.001
5 or more	2.58	2.20-3.02	<0.001
Number of times smoked cigarettes in last month			
None	1 (reference)		
1-60	1.45	1.24-1.70	<0.001
61 or more	1.90	1.62-2.23	<0.001
Number of times used cannabis in last 30 days			
None	1 (Reference)		
1-10	1.56	1.34-1.81	<0.001
11 or more	1.92	1.58-2.34	<0.001
Age (centered at 16)	0.74	0.73-0.77	<0.001
Age squared	1.01	1.00-1.01	<0.001
Number of drinks X age			
0	(Reference)		
1-4	0.98	0.96-1.00	0.932
5 or more	1.05	1.02-1.07	<0.001
Ethnicity			
White	(Reference)		
Black	1.62	1.44-1.82	<0.001
Asian	1.10	0.90-1.32	0.482
Other	1.50	1.27-1.73	<0.001
Gender (female)	0.40	0.36-0.43	<0.001
Delinquency	1.20	1.18-1.22	<0.001
IQ	0.98	0.98-0.98	<0.001
Temper	1.25	1.15-1.37	<0.001
Depression	1.01	1.01-1.02	<0.001
Parents argue	1.18	0.99-1.42	0.070
Peer substance use	1.24	1.17-1.32	<0.001
Neighbourhood violent crime rate	1.02	1.00-1.02	<0.001
Neighbourhood disadvantage	1.09	1.04-1.14	<0.001
Alcohol X delinquency			
0	1		
1-4	0.96	0.94-0.98	<0.001
5 or more	0.95	0.93-0.97	<0.001
Cigarettes X delinquency			
None	1		
1-60	0.99	0.96-1.00	0.233
61 or more	0.95	0.90-1.09	0.839
Cannabis X delinquency			
No	1		
Yes	0.97	0.95-0.98	0.001

Figure 27 shows the marginal effect of the number of drinks consumed on the predicted probability of group fighting, and shows a similar patter to that found with other measures of violence. Those in the highest alcohol consumption category had a higher risk of violence in a group than non drinkers throughout. Those who drank 1-4 drinks each occasion had a similar risk of violence to those who drank 5 or more drinks at age 12. The risk then decreases at a faster rate than the heaver drinkers and converges with the trajectory for the non-drinkers by age 24. This pattern was predicted in both males and females (see Figure 28).

Figure 27. Predictive marginal effects of number of drinks consumed on probability of group fighting

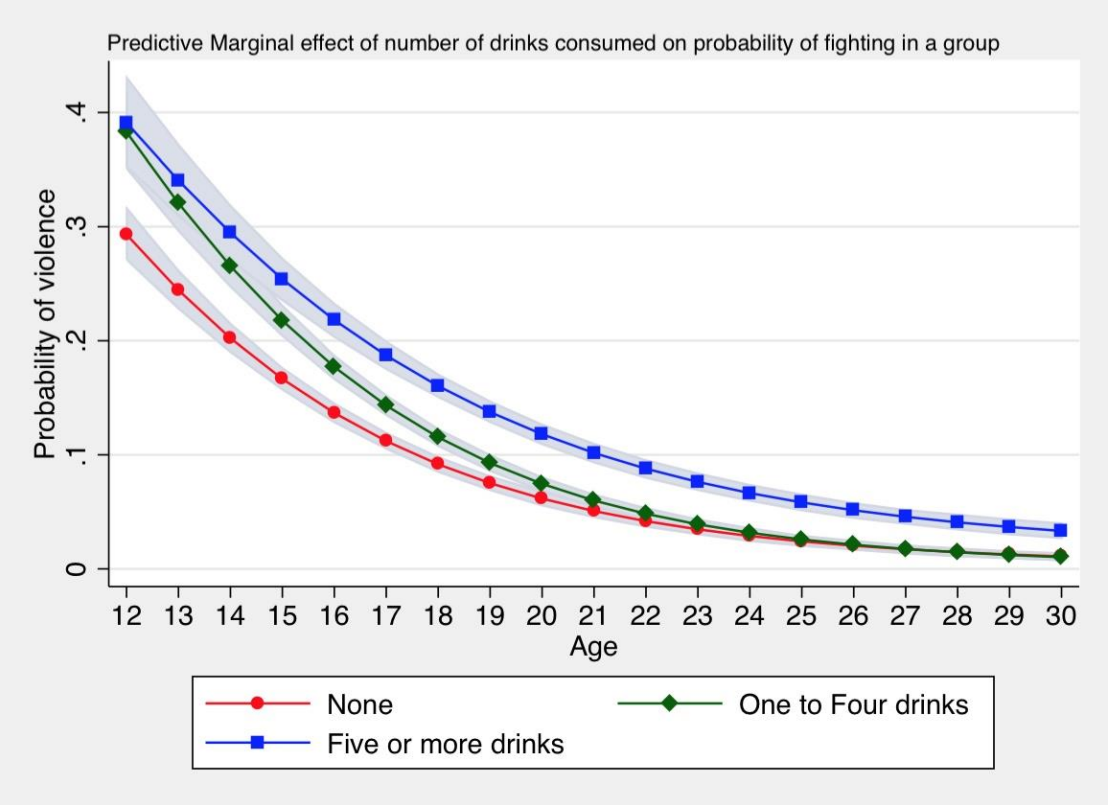
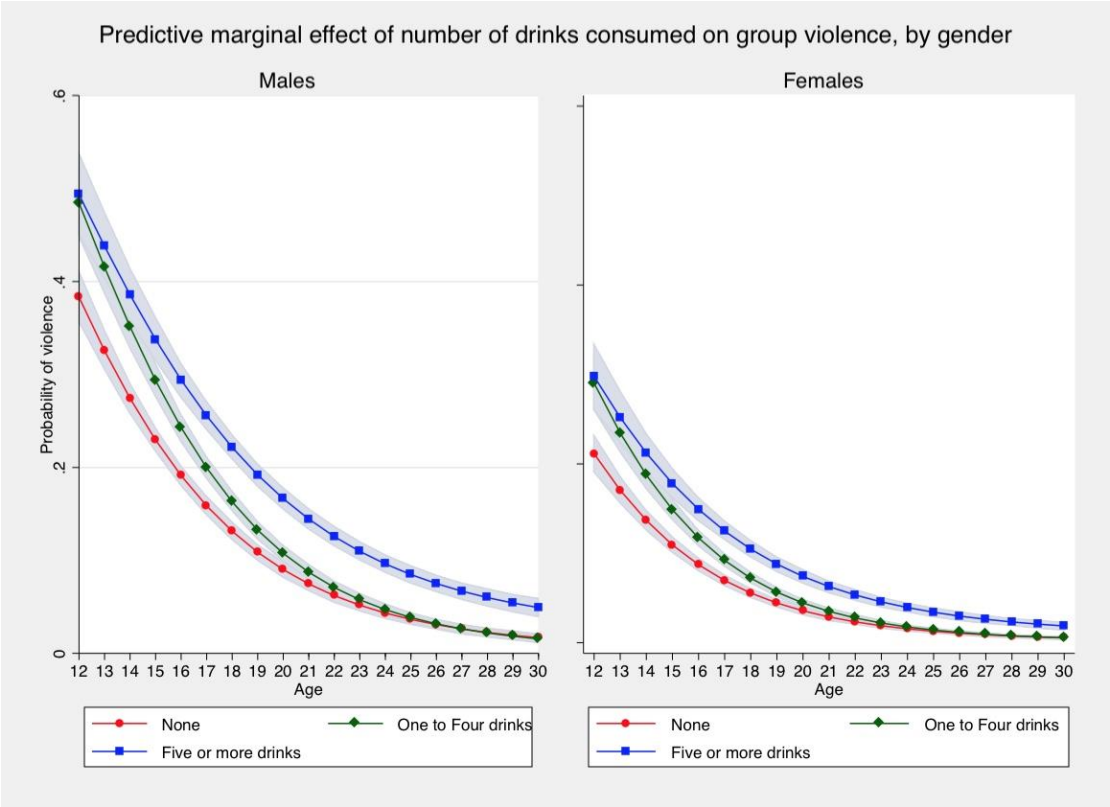


Figure 28. Predictive marginal effect of number of drinks consumed on probability of fighting in a group, by gender



CHAPTER 15 RELATIONSHIP BETWEEN ALCOHOL, PERSONALITY FACTORS AND VIOLENCE

DESCRIPTIVE STATISTICS

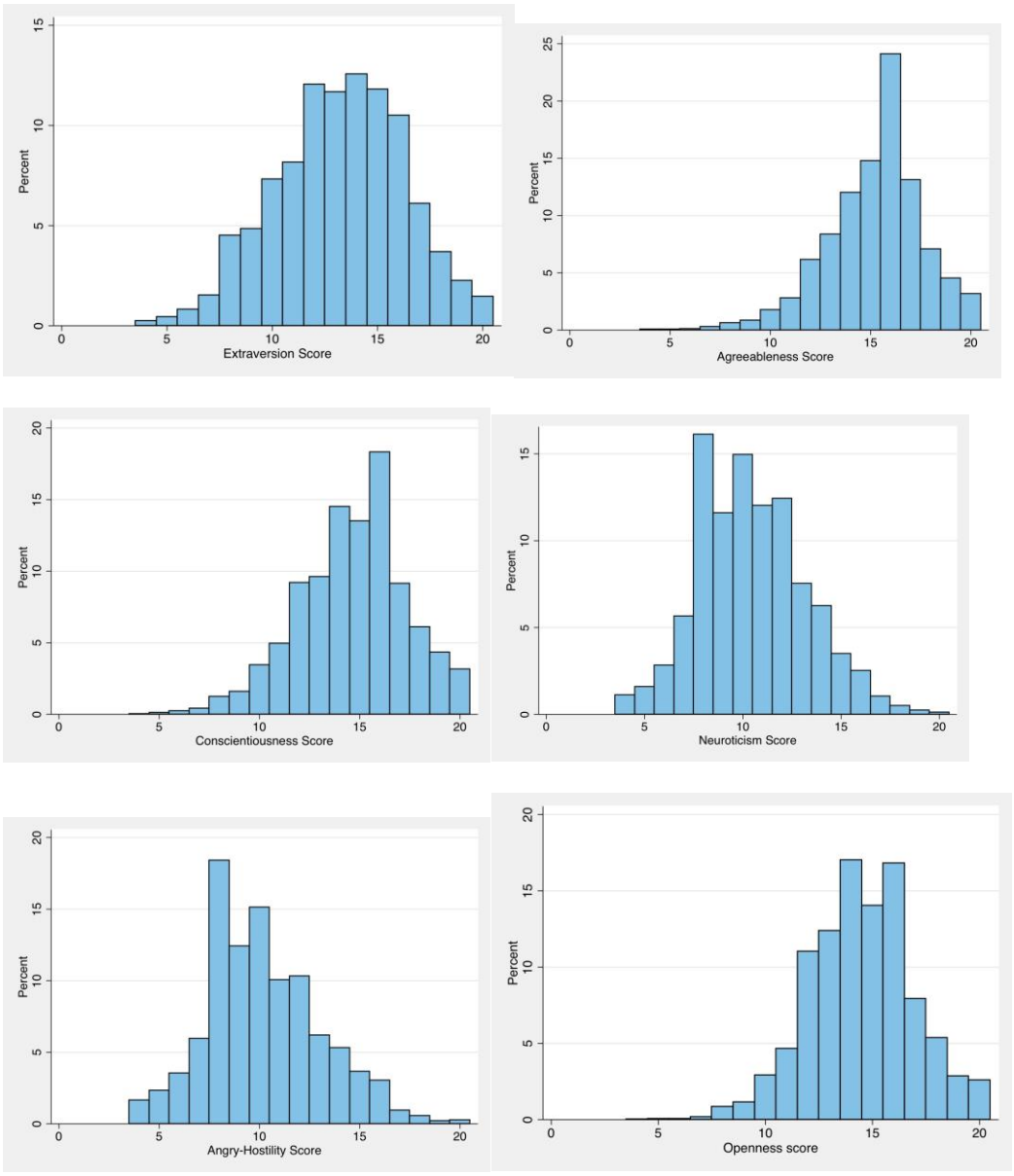
All analyses were conducted on all participants at wave IV. The mean age of the participants was 29.0 years (SD 1.75). The median and inter-quartile range of each personality item is shown in Table 41, and shows similar scores among males and females. For the purpose of preliminary analyses, scales for each personality factor were derived by combining scores from the relevant items, yielded scales that were approximately normally distributed. The mean and standard deviations for each scale were as follows: Extraversion (13.2, 3.1), Agreeableness (15.2, 3.1), Conscientiousness (14.6, 2.4), Neuroticism (10.4, 2.7), Openness (14.5, 2.5).

In total 1,214 (7.7%, 95% CI 7.3-8.2) engaged in at least one violent act in the past 12 months before wave IV. In total, 4,144 (75%) of men and 5677 (68%) of females drank alcohol at least once in the year prior to interview (χ^2=110, p<0.001). The mean number of drinks consumed each occasion for men was 3.3 (SD=3.4), and for women was 2.4 (SD=2.4, t=27.1, p<0.001). A total of 4044 (55%) of men and 3339 (40%) of women engaged in binge drinking at least once over the year prior to interview (χ^2=349, p<0.001), and similar proportions reported getting drunk at least once (56% of men compared with 40% of women, χ^2=389, p<0.001). The data from men and women were combined for further analyses.

Table 41. Unadjusted relationship between personality factors and violence (odds ratios with 95% confidence intervals)

	Mean (SD)	OR	95% Confidence Interval	p
Extraversion	13.2 (3.1)	1.05	1.01-1.09	0.020
Agreeableness	15.2 (2.4)	0.85	0.81-0.89	<0.001
Conscientiousness	14.6 (2.7)	0.93	0.89-0.97	<0.001
Neuroticism	10.4 (2.7)	1.11	1.06-1.15	<0.001
Angry-Hostility	10.2 (2.9)	1.20	1.16-1.25	<0.001
Openness	14.5 (2.5)	1.06	1.01-1.11	0.015

Figure 29. Frequency distributions of personality factor scales



CONFIRMATORY FACTOR ANALYSIS

Applied to data used in the current study, Anger-hostility score was found to correlate highly with neuroticism (0.69), but not with the other traits(-0.12 to -0.16). Anger-hostility is considered to be a "facet" or sub-component of neuroticism (Costa and McCrae, 1995) and therefore the anger-hostility factor was defined as a second order factor indicated by the latent factor neuroticism and the 4 anger-hostility items.

A confirmatory factor analysis was carried out to determine whether each of the variables loaded onto their respective latent constructs. All of the factors (Extraversion, Agreeableness, Conscientiousness, Neuroticism, Openness, Anger/Hostility Alcohol and Violence) were each measured by 4 variables. In the initial analysis, all factors loadings were allowed to vary freely except for the first measure of each factor which was constrained at 1.0 to identify the metric of the latent variable. All factor intercorrelations were freed, as were the error terms within the same measure.

The initial model did not fit the data very well (CFI=0.90, TLI=0.89, RMSEA=0.056, WRMR=6.62, χ^2=27391, p<0.001). Model fit was improved by specifying correlations between personality variables guided by the model fit indices. The resulting model fitted the data reasonably well (CFI=0.95; TLI=0.94; RMSEA=0.044, 95% CI = 0.043-0.045; WRMR=4.64; χ^2=13,250, p<0.001). A significant χ^2 was expected as the test is sensitive to sample size. As another check for similarity between the sexes and of internal consistency of the model, the confirmatory factor analyses were run separately for men and women with almost identical model fit in each case. The standardised factor loadings for each variable are shown in Table 42 and shows acceptable loadings onto respective factors.

STRUCTURAL MODELS

As shown in Table 43 Anger-Hostility was significantly associated with alcohol use; the standardised coefficient was 0.37. This means that for an increase in Anger-Hostility by 1 standard deviation, alcohol use increases by over one third of a standard deviation. Anger-hostility was also associated with cannabis use (standardised estimate 0.14), but not with smoking.

The direct and indirect effects of personality factors, alcohol, cannabis and cigarette smoking is shown in Table 44 there were significant direct effects of alcohol use, cannabis use and cigarette smoking on violence. The standardised estimate for the direct effect of alcohol on violence was 0.12 (95% CI 0.09, 0.15). This means that as alcohol use increases by 1 standard deviation, violence increases by around 12% of a standard deviation. A similar finding was observed for smoking (standardised estimate 0.12, 95% CI 0.09, 0.14), and a smaller but significant effect was observed

for cannabis (standardised estimate Anger-Hostility was strongly associated with violence.

Extraversion was positively associated with violence and alcohol, and to a lesser extent cigarette smoking and cannabis use. The standardised coefficient for the effect of extraversion on alcohol was 0.24, and for cigarette smoking was 0.11 and 0.07 respectively. The total standardised effect of extraversion on violence was 0.18 (0.14, 0.22). Approximately 16% of the effect of extraversion on violence was mediated by alcohol, approximately 7% mediated by smoking, and approximately 3% by cannabis use.

Agreeableness was inversely associated with violence, alcohol use, smoking and cannabis use (see Table 43 and Table 44). The standardised effect of agreeableness on violence was -0.33 (95% CI -0.38, -0.27), meaning that as agreeableness reduces by 1 standard deviation, violence increases by one third of a standard deviation. Approximately 8% of the effect of agreeableness on violence is mediated by alcohol. Around 3% of the effects are mediated by each of cannabis use and cigarette smoking.

Conscientiousness was also inversely associated with violence, alcohol, cigarette smoking and cannabis use, although the effects were small. The standardised effect of conscientiousness on violence was -0.05 (95% CI -0.09, -0.01), indicating that violence increases by around 5% of a standard deviation for every 1 standard deviation decrease in conscientiousness. However, alcohol and smoking each contributed around 20% of the total effect, and cannabis contributed 8%.

Neuroticism was inversely associated with alcohol and violence. It was not associated with either cigarette smoking or cannabis use. The total effect of Neuroticism on violence was -0.37, indicating that for every standard deviation reduction in Neuroticism, violence increases by one third of a standard deviation. Approximately 11% of the effect is mediated by alcohol.

Openness was associated with alcohol use (standardised estimate 0.22), and cannabis use (standardised estimate 0.19), and to a lesser extent, cigarette smoking (standardised estimate 0.04). It was also significantly associated with violence (standardised estimate 0.23, indicating that violence increases by almost a quarter of a standard deviation for every standard deviation increase in openness).

Approximately 11% of the effect of openness on violence is mediated by alcohol, 7% is mediated by cannabis use, and 2% by smoking.

The proportion of the variance of violence explained by the model (R^2) was 23.2%

ALTERNATIVE MODELS

Theory would suggest that personality factors underpin alcohol use and aggression, however alternative structural models were tested in which personality factors were specified as mediating the relationship between alcohol and violence. Model fit was poorer than that for the primary models tested (CFI=0.89, TLI=0.88, RMSEA=0.065 (95% CI 0.064-0.066,), WRMR=5.29, χ^2=14,691, p<0.001) for men, and for women CFI=0.86, TLI=0.83, RMSEA=0.069 (95% CI 0.068-0.070,), WRMR=6.09, χ^2=18,771, p<0.001.

Table 42. Items used to indicate latent factors: Median and interquartle ranges and standardised factor loadings

Variable	Median (IQ range)	Standardized factor loadings
Extraversion		
Life of the party*	3(3-4)	0.39
Don't Talk a lot	2(2-4)	0.63
Talk to a lot of different people at parties*	4(3-4)	0.66
Keep in the background	3(2-4)	0.79
Agreeableness		
Sympathize with others' feelings*	4(4-4)	0.45
Not interested in other's problems	4(3-4)	0.64
Feel other's emotions*	4(3-4)	0.55
Not really interested in others	4(3-4)	0.94
Conscientiousness		
Gets chores done right away*	4(3-4)	0.33
Often forgets to put things back in their proper place	4(2-4)	0.54
Likes order*	4(3-4)	0.36
Makes a mess of things	4(3-4)	0.97
Neuroticism		
Frequent mood swings *	2(2-3)	0.65
Relaxed most of the time	2(2-3)	0.44
Get upset easily*	2(2-3)	0.88
Seldom feels blue	2(2-4)	0.28
Angry-Hostility		
Gets angry easily*	2(2-3)	0.87
Rarely gets irritated	3(2-4)	0.63
Loses temper*	2(2-3)	0.66
Keeps cool	2(2-2)	0.79
Neuroticism		0.92
Openness		
Has a vivid imagination*	4(3-4)	0.40
Not interested in abstract ideas	3(3-4)	0.57
Has difficulty understanding abstract ideas	4(3-4)	0.55
Does not have a good imagination	4(4-5)	0.65
Violence		
How often in a serious physical fight	0 (0-0)	0.95
How often in a fight where a group of your friends was against another group?	0 (0-0)	0.84
How often hurt someone badly enough to need bandages or care from a doctor or nurse?	0 (0-0)	0.98
How often pulled a knife or gun on someone?	0 (0-0)	0.49
Alcohol		
Frequency of drinking alcohol	3(1-4)	0.82
Number of drinks usually consumed	3(1-5)	0.66
How many days drank drink 5 or more drinks in a row	1(0-3)	0.88
How many days intoxicated with alcohol	1(0-2)	0.87

* Indicates that the scoring is reversed
All factor loadings are significant at p<0.001

Table 43. Standardised coefficients of personality factors on alcohol, cannabis use and cigarette smoking

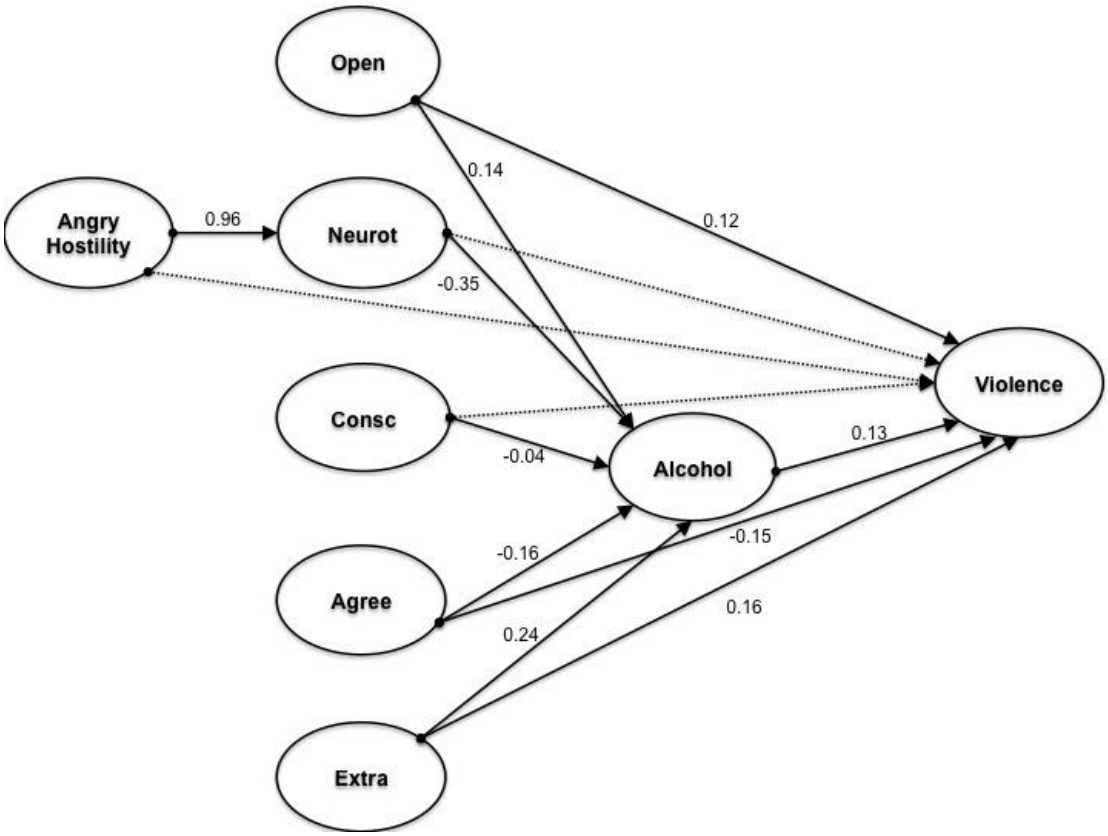
	Alcohol	Smoking	Cannabis
	Std Coef	Std Coef	Std Coef
Extraversion	0.24**	0.11**	0.07**
Agreeableness	-0.22**	-0.09**	-0.14**
Conscientiousness	-0.09**	-0.10**	-0.05**
Neuroticism	-0.36**	0.07	-0.05
Anger-Hostility	0.37**	0.03	0.14**
Openness	0.22**	0.04*	0.19**

Table 44. Standardised coefficients of personality factors, alcohol, cannabis and cigarette smoking on violence

	Violence		
	Estimate	**SE**	**95% CI**
Extraversion			
Direct effect	0.133**	0.028	0.088, 0.178
Indirect via alcohol	0.029**	0.005	0.021, 0.037
Indirect via cannabis	0.006**	0.001	0.003, 0.008
Indirect via smoking	0.012**	0.002	0.009, 0.016
Total effect	0.180**	0.027	0.136, 0.224
Agreeableness			
Direct effect	-0.279**	0.030	-0.328, -0.230
Indirect via alcohol	-0.026**	0.005	-0.034, -0.018
Indirect via cannabis	-0.012**	0.002	-0.015, -0.008
Indirect via smoking	-0.010**	0.002	-0.014, -0.007
Total effect	-0.327**	0.030	-0.375, -0.279
Conscientiousness			
Direct effect	-0.025	0.024	-0.064, 0.014
Indirect via alcohol	-0.011**	0.002	-0.014, -0.007
Indirect via cannabis	-0.004**	0.001	-0.007, -0.002
Indirect via smoking	-0.011**	0.002	-0.015, -0.008
Total effect	-0.051*	0.024	-0.090, -0.012
Neuroticism			
Direct effect	-0.334**	0.094	-0.488, -0.180
Indirect via alcohol	-0.043**	0.009	-0.058, -0.028
Indirect via cannabis	-0.004	0.004	-0.010, 0.002
Indirect via smoking	0.009	0.006	-0.001, 0.018
Total effect	-0.372**	0.093	-0.525, -0.219
Anger-Hostility			
Direct effects	0.535**	0.094	0.380, 0.689
Indirect via alcohol	0.045**	0.009	0.030, 0.060
Indirect via cannabis	0.011**	0.004	0.005, 0.018
Indirect via smoking	0.003	0.005	-0.005, 0.012
Indirect via neurot	-0.308**	0.087	-0.451, -0.165
Total effect	0.250**	0.021	0.216, 0.285
Openness			
Direct effects	0.188**	0.034	0.132, 0.243
Indirect via alcohol	0.026**	0.005	0.018, 0.034
Indirect via cannabis	0.016**	0.003	0.011, 0.020
Indirect via smoking	0.005**	0.002	0.001, 0.008
Total effect	0.234**	0.032	0.181, 0.287
Direct effect alcohol	0.120**	0.020	0.086, 0.153
Direct effect cannabis	0.083**	0.014	0.060, 0.106
Direct effect smoking	0.116**	0.016	0.090, 0.143

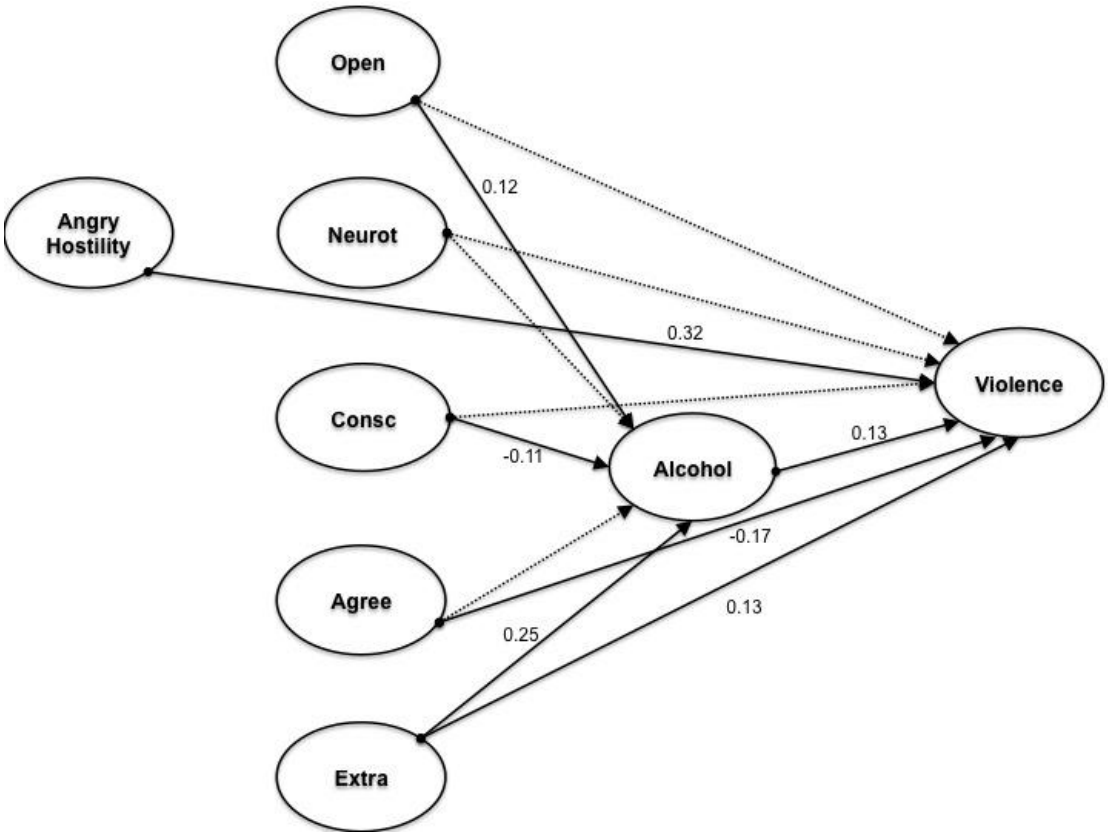
SE, Standard Error; CI, Confidence Interval. *p<0.05, **p<0.01

Figure 30. Final model of standardized effects of personality factors on alcohol and violence: men



Only effect sizes p<0.05 are shown. Correlations between latent factors are not shown.

Figure 31. Final model of standardized effects of personality factors on alcohol and violence: women



Only effect sizes p<0.05 are shown. Correlations between latent factors are not shown.

SECTION IV DISCUSSION

CHAPTER 16 ASSOCIATION BETWEEN ALCOHOL USE AND VIOLENCE

SUMMARY OF FINDINGS

There is evidence of a relationship between heavy alcohol consumption during early teenage years and risk of initiation of violence during the year following this. Those adolescents who drank 5-10 drinks each occasion were twice as likely to report serious violence one year later than those who did not drink after controlling for individual, family and neighbourhood factors, however drinking 1-4 drinks each occasion was not associated with violence. There was evidence of a linear trend between number of drinks consumed and violence. There was no evidence that frequency of alcohol consumption was related to interpersonal violence, or indeed that the overall quantity of alcohol consumed was associated with violence. This implies that heavy episodic drinking is associated with the initiation of serious violence in adolescence. This is further evidenced by the finding of a linear relationships between both the frequency of binge drinking, and the frequency of getting very drunk and violence initiation. Furthermore, when both are entered simultaneously into a model the number of drinks consumed, but not the frequency of use is associated with the initiation of violence.

Estimation of the number needed to prevent found that, assuming there was no residual confounding, it would be necessary to prevent 47 people (CI 25-146) from binge drinking to prevent one from becoming violent within the next year.

When the analyses were extended to include initiation of violence at any point between wave II and IV, there was again strong evidence for a relationship between alcohol consumption and violence. Evidence was weaker for a linear relationship between frequency of drinking and violence. There is therefore strong evidence that quantity of alcohol rather than frequency of consumption is associated with the initiation of violence.

Longitudinal models including the entire cohort and taking into account changes in the amount of alcohol reportedly consumed showed strong evidence of a relationship between alcohol consumption and violence; overall, drinking 1-4

drinks each occasion was associated with a 36% increase in risk of violence, and for those consuming 5 or more drinks, the risk increased by 214% compared with those who did not drink alcohol.

The effect was dynamic with the findings indicating that those who drank 1-4 drinks had an elevated risk of violence only during adolescence (not adulthood) compared with those who did not drink. Furthermore, for those who drank 5 or more drinks, the risk was highest amongst adolescents, but the relative risk gradually reduced and tended to converge by the 4th decade. Similar patterns were found for both males and females.

The effect of alcohol on risk of serious fighting and of serious violence was very similar, however, there were differences in the effect of alcohol on fighting in a group. The effect of heavy drinking on fighting in a group was greater, and this effect, continued to be evident until the 4th decade. The effect also remained significantly higher compared with those who drank more moderately, or did not drink at all, particularly in males.

EXPLORATION OF CAUSAL INFERENCE

There is evidence that alcohol and violence are associated, but in addition to causality there are other possible explanations. They are that violence may cause alcohol consumption rather than the other way around (reverse causality), that there are systematic errors in the study that result in an incorrect estimate of the association (bias), that the observed association is due (totally or in part) to the effects of one or more other variables (confounding), or that the association is found by chance. I will explore these possibilities in turn.

REVERSE CAUSALITY

In both the crude and adjusted analyses, alcohol was associated with violence. It is possible however that the observed association between alcohol and violence is due to reverse causality, that is that people who are violent subsequently misuse alcohol.

It is known that some offenders who commit violent acts are traumatised by their own violence (Evans *et al.*, 2007), and that exposure to violent trauma may lead to an increase in alcohol consumption (Kelley *et al.*, 2013), and that it is therefore possible that violent people drink alcohol to self-medicate (Khantzian, 1985).

It is also the case that, developmentally, aggression precedes the initiation of alcohol use; aggression is a normal human behaviour in infancy but in most children it reduces by the time they enter primary school (Tremblay *et al.*, 2004). A minority of children do not grow out of their aggression, and there is evidence that those who are still displaying violence during adolescence are more likely to be aggressive as adults (Broidy *et al.*, 2003, McCord *et al.*, 2001). Therefore, although aggression is normal in early childhood and developmentally precedes the initiation of alcohol use, most children largely grow of aggression as they learn to control it, but those who do not, tend to continue to be aggressive in adulthood, and it is possible that these individual are more likely to misuse alcohol.

As stated in the introduction however, there is little evidence from longitudinal studies that violence is associated with later alcohol use (for example evidence from the Seattle Social Development Project found no evidence that aggression predicted later alcohol use (Huang *et al.*, 2001)). As stated in the introduction, there is however some evidence that alcohol use predicts later aggression (for example, (Dubow *et al.*, 2008)). The aims of my study were to investigate the effect of alcohol on violence, and therefore to reduce the likelihood of reverse causality in this study, firstly a longitudinal design was used in which the measurement of alcohol exposure preceded the measurement of violence. To further reduce the likelihood of reverse causality, as explained in the methods section analyses in Chapter 12, the first set of analyses were carried out after excluding those adolescents who were violent at baseline, and therefore reduced the likelihood of the results being due to reverse causality.

BIAS

SELECTION BIAS

Selection bias occurs when the method of selection of the participants is systematically different from the population of interest. In this study, as explained in the methods section, participants were selected using a stratified randomised

design from schools across the whole of the USA. A sample weight was calculated for each participant to account for the probability of selection and, in my study, in the analyses that investigated incident violence (Chapter 12) statistical methods were used which took account of the study design, and incorporated the study sample weights to ensure that the estimates were unbiased and nationally representative. It is therefore unlikely either the participant selection or analysis of the data has biased these results. It was not possible to incorporate sample weights into my analyses of dynamic change in exposure and outcome (Chapter 13) because of the software used for analysis, however there is no reason to suspect that the failure to incorporate sample weights would have significantly biased the results.

MISSING DATA AND CASES LOST TO FOLLOW-UP

Loss to follow-up is inevitable in most cohort studies and can introduce bias and loss of statistical power. There are no agreed standards on acceptable follow-up rates, however it has been suggested that 60% us adequate, 70% is good and 80% is very good (Babbie 1973). Perhaps more important than the percentage of cases lost to follow-up is the mechanism by which the follow-up data has been lost. There are three mechanisms that have been described: missing completely at random (MCAR), missing at random (MAR), and missing not at random (MNAR) (Little and Rubin, 1987). Participants who drop out of the study for reasons that are independent of the exposures, confounders and outcome are considered MCAR. Therefore loss to follow-up in this situation would not introduce bias, and would only reduce the statistical power to detect the association of interest. Observations that are MAR are those in which the loss to follow-up depends on the exposures or confounders, but not the outcome. Missing information due to MCAR and MAR are considered to be "ignorable" because the collected variables can be used to adjust for the potential bias using multivariate analysis (Schafer and Graham, 2002). More problematic, are observations that are MNAR, which are those in which dropping out of the study is related to the outcome, but can not be explained by the observed variables. In cohort studies, loss to follow-up is often MNAR.

A statistical simulation study in a cohort of 500 observations showed that when missing observations with either MCAR or MAR, with as much as 60% of the observations missing there was no significant bias. However, observations that were lost due to the MNAR mechanism caused increasingly biased estimates as the

proportion of missing data increased, particularly when the proportion of observations lost exceeded 20%(Kristman *et al.*, 2004).

At Wave I in Add Health, there was very little missing information on any of the questions. Out of over 17,000 individuals, only 36 (0.21%) failed to provide a response on how frequently they drank alcohol, and 156 (0.89%) failed to give a response to questions about perpetration of violence. There was attrition of the sample after the first wave. Wave II achieved an 88.6% response rate, wave III a 77.4% response rate, and wave IV an 80.3% response rate. As shown in the simulation study above, bias can be introduced particularly when the proportion of missing data exceeds 20%; in the current study, the amount of missing data was less than 20% and the risk of bias introduced is considered to be at an acceptable level.

As stated in the methods (page 90-91), missing data was adjusted for to some extent by including the sample weights in the analyses. As described in the methods, the sample weights use a method known as Inverse Probability Weighting to adjust for individuals who are missing at follow up. The principle is that individuals who had a high probability of dropping out are given a higher weighting to adjust for those who had as similar characterises but did drop out. The disadvantage of this method is that baseline characteristics may not be very reliable as predictors of follow-up and that drop-out may be related to the dependent variable of interest, which this method can do nothing to resolve.

Another method for dealing with missing data is to impute the missing data, also by creating a model using baseline characteristics. A popular method is know as multiple imputation, first proposed by Rubin (Rubin, 1977). This is a method in which multiple "complete" datasets of plausible values are produced; each dataset has imputed missing values that incorporates random variation. The analyses are the carried out across the multiple datasets to produce a single estimate. This method introduces random error into the imputation and therefore appropriate standard errors.

Some limitation in applying this method to survey data include mis-specification of the model used for imputation, violations of the assumption that the data are missing at random (MAR), and problems of non-convergence of the models, particularly when there are many predictors (White *et al.*, 2011). Nevertheless, multiple imputation is a valuable method when used appropriately. This method

was not adopted for two reasons. Firstly it was felt that the survey weights produced by IPW provided a sufficient adjustment for missing data. Second, simulation studies have shown that there is only likely to be a low risk of bias when the amount of missing data is less than 20%, and thirdly, there were practical reasons why multiple imputation could not ne used in this study. Stata, the statistical software used for this study does not have the facility to analyse multiple datasets as produced in multiple imputation in the random-effects models.

Attrition in this study could have resulted in an over-estimate of the association between alcohol and violence only if drinkers who became violent were less likely than drinkers who did not became violent to drop out, or if those who were non-drinkers who did not become violent were more likely to drop out. It is more likely however that those who were violent, and drinking alcohol were more likely to engage in other problematic or chaotic behaviours, and thus less likely to be traced or to participate in follow-up. It is possible therefore that, if anything, the extent of the relationships may be underestimated. That said, the Add Health investigators conducted interviews amongst those located in institutions, including prisons to reduce the risk of bias. In addition, the calculation of sample weights for each wave adjusted for the probability of inclusion in the study, thus adjusting for those who had dropped out.

The random effects analysis (Chapter 14) modelled change in exposure (alcohol consumption) over time and measured the extent to which this related to change in violence. Data from all individuals who participated in two or more, not necessarily consecutive, waves of data collection were included in the study to maximise the information available, thus mitigating against non-participation in one or two waves.

The extent to which attrition may have affected the observed relationship between alcohol and violence cannot be known, however given the acceptably low attrition rate and the methods used for statistical analysis, it is unlikely that the observed results were affected to any great extent by missing data, or study attrition.

INFORMATION BIAS

Reporting and Recall Bias

There is a possibility that information collected differently between two groups can lead to an error in the conclusion drawn from the observed association.

All of the measures rely on self-report and there is the possibility that either recall or reporting bias may be introduced. For example, those who are seriously violent may be more likely to over-estimate their alcohol use, and that those that drink heavily may be more likely to over-estimate their violence (recall bias), hence explaining at least part of the association. The other possibility is that individuals who are violent are more likely to seek to portray themselves as heavy drinkers and vice versa (reporting bias).

Self-reported data is however commonly used in research of this kind, as it provides the opportunity to obtain far more detailed information that that which is generally available in official records, and tends to be more complete than other sources of information such as hospital records, or official arrest or conviction data (Elliott *et al.*, 1989). In addition the methods used (Audio Computer-Assisted Self-Interviewing) has been shown to increase valid and accurate reporting in comparison with face to face interviews with researchers, (Turner *et al.*, 1998b) thus reducing the likelihood of reporting bias. Farrington and colleagues also investigated the validity of self-reported delinquency by comparing self-reports of arrests and convictions to both a combined scale comprising reports from parents, teachers and self-report, and with official records and found high concurrent validity for self-reported delinquency (Farrington *et al.*, 1996).

Overall, the prevalence of alcohol use and misuse in this study is similar to that found in other national surveys in the USA and is therefore unlikely to have suffered from substantial misreporting. The National Institute of Alcohol Abuse and Alcoholism (NIAAA) in the United States of America reported trends of alcohol use among young people age 12-20 annually between 1991 and 2011(Chen *et al.*, 2013). The most direct comparison with data from Add Health can be made between NIAAA data for 15-17 year olds in 1996 and wave II Add Health data that was carried out in 1996 when the median age of the sample was 15.9. The NIAAA survey showed that the mean frequency of drinking among 15-17 year olds was approximately 5 days in the previous 30. The mean number of drinks consumed each occasion among was 4.1, compared with a median of 4 drinks in Add Health. The NIAAA survey reported that 12.3% of 15-17 year olds had engaged in binge drinking in the previous 30 days. This is somewhat lower than the 20% in the Add Health survey who reported that they drank once a month or more. However binge drinking increases markedly with age; in the NIAAA survey , 28% of 18-20 year olds reported they had been binge drinking in the previous month. Given that the

age range for participants in Add Health at Wave II included some older participants (up to age 19) than those in the NIAAA survey , it is to be expected that the median frequency of binge drinking may be slightly higher.

The NIAAA survey reported frequency of having been "drunk or very high from drinking alcoholic beverages", wording very similar to that used in Add Health, however the former enquired responses to the question over the preceding 30 days, whereas Add Health covered the previous year. Around 30% in the Add Health study reported getting very drunk in the past year, and around 17% in the NIAAA survey reported having done so in the preceding 30 days, and therefore a higher proportion would be expected over the longer time-frame of enquiry.

Overall, it appears that the prevalence and patterns of drinking reported in the Add Health study are similar to those in other studies, and therefore unlikely to be subject to significant misreporting.

Misclassification Bias

There is a risk of misclassification of the violent and non-violent group at baseline, such that some people who reported no violence within the previous twelve months, but who had been violent prior to that were erroneously classified as non-violent. It is possible that some individuals were not excluded who were otherwise aggressive, but who had not yet engaged in any acts of serious violence. The overall trend however throughout the study was towards a reduction in rates of violence. Participants were at greatest risk of violence at the first wave and therefore excluding those who were violent at wave I is likely to have excluded the vast majority of people who were dispositionally violent. The inclusion of many violent people, misclassified as non-violent would have the effect of increasing the apparent association between alcohol and violence. However, as stated above, there is ample evidence from the literature of developmental continuity in violence, and that most people who had been violent (unrelated to alcohol) were likely to have been so within the past 12-months before inception into the study.

There was a sharp decline in reported violence between wave I and II; approximately 14% of individuals who reported serious violence at wave I reported no violence at wave II. It is possible that the reason for the sharp decline in reported violence between the two waves was due to misclassification of violence at wave I (over reporting) due to individuals exaggerating their violence,

or perhaps that individuals did not understand the question. By comparison, I found broadly similar reductions in violence between wave I and II in the secondary measures of violence, serious fighting (19%) and fighting in a group (11%), indicating that there is little evidence that the specific question chosen as the primary violence outcome measure was specifically misunderstood. There was however evidence that the younger the individual, the more likely they were to report a reduction in violence. An exploratory logistic regression to investigate factors that predicted a fall in violence between wave I and II in a model including all of the independent variables used in the models to investigate substance use and violence in this thesis found that age was a significant predictor (OR 0.91, 95% CI 0.88-0.95). Other significant predictors were delinquency (OR 1.1, 95% CI 1.10-1.12), gender (OR 0.4, 95% CI 0.38-0.78), ethnicity (black compared with white OR 1.48, 95% CI 1.27 – 1.72), IQ, (OR 0.8, 95% CI 0.76-0.88), depression (OR 1.01, 95% CI 1.00-1.02) and temper (OR 1.51, 95% CI1.35-1.70). As age was a significant predictor of a fall in violence, it is possible this was due to a misclassification of violence among the younger respondents at wave I. However there was no evidence that was no evidence of a general tendency to exaggerate deviant behaviour in general among the youngest in the sample as this was not seen in reports of smoking, drinking or cannabis. There remains the possibility however that the younger participants over-reported violence specifically, and this would have the potential of introducing bias. Nevertheless it should be noted that all of the analyses in chapters 12 and 13 were carried out on the group of people who reported no violence at wave I.

There is a possibility that those who had perpetrated violence were not captured by the questions asked at each of the waves. The questions used to measure violence asked about violence within the past 12-months. There is a possibility that individuals who had engaged in violence more than 12-months before the questionnaire (or were recalled by the participants of having occurred more than 12-months before) were misclassified as being non-violent. Bias would be introduced only if this occurs systematically more or less in either drinkers or non-drinkers. It is impossible to know whether this occurred, but there is no evidence to suggest that it did. At Wave II, this is unlikely to have occurred as the interviews took place approximately 1 year after the wave I data collection, and therefore incorporated the entirety of the follow-up period. Waves III and IV were separated by several years, and would not have captured violence that took place more than a

year prior to each wave interview. Neither would it have captured changes in levels of alcohol use more than a year prior to each wave. It is therefore possible that there has been an under-estimation of violent incidents during the entirety of the follow-up period, but this is likely to have occurred in drinkers and non-drinkers.

The non-violent group was defined as those who reported no violence within the past 12 months at Wave I. Ideally, it would have included all those who had never engaged in serious violence. Therefore some individuals could have engaged in violence more than 12-months prior to their interview at Wave I, and therefore some individuals could have been misclassified as having initiated violence at wave II when in fact they had already initiated violence. As others have noted however, violence tends to be relatively stable across time (Farrington, 1989, White *et al.*, 1993), and, indeed as this study and others have shown (e.g. (Duncan *et al.*, 1997, Moffitt, 1993) tends to reduce over time. There is evidence therefore to suggest that overall it is unlikely that individuals would have been misclassified in this way.

In summary, there are risks of missclassifcation of violence, they are small and unlikely to affect the exposure groups differentially.

Overall, although it cannot be known, it is not very likely that misclassification has introduced significant bias in this study.

CONFOUNDING

The association between alcohol use and violence was adjusted for several factors that were, *a priori*, known to be associated with alcohol misuse and violence. An extensive set of factors including individual, family, and neighbourhood characteristics were adjusted for. An additional strength was the ability to control for official rates of violent crime in the local community, as well as official indicators of deprivation.

By far, the largest effect on the relationship between alcohol use and violence was observed after adjusting for age at Wave I; there was an increase in the observed association by 84% once adjusted for age. Younger people were less likely to drink alcohol, but there was a higher rate of violence among those who drank at an

earlier age compared with those who were older and drank alcohol. Other studies have shown that the rates of alcohol consumption generally increases during adolescence and young adulthood(Hingson *et al.*, 2001), whereas the rates of violence generally reduce over time, therefore this could have obscured the true relationship between alcohol and violence.

IQ is positively associated with alcohol consumption, but negatively with violence. Adjustment for IQ increased the observed association by around 13%. By contrast, there were several factors that reduced the apparent association once they were controlled for. Two variables were responsible for large confounding effects; they were peer drug use, and delinquency which both reduced the apparent association by over 70%. As stated in Chapter 3 there is a theory that delinquency, violence and substance misuse are part of a problem behaviour syndrome, and that each of these behaviours may be expressions of a common underlying phenotype. As explained in Chapter 3 however, although there is a strong association between these factors, there is some prior evidence that a single common factor cannot adequately explain both substance use and delinquency (Osgood *et al.*, 1988, Paradise and Cauce, 2003, White and Labouvie, 1994). A partial explanation may lie in the evidence that, although violent and non-violent delinquency are strongly associated (Lipsey *et al.*, 1997), there are different and distinctive pathways for violent and non-violent offending (LeBlanc and Loeber, 1998, Tremblay *et al.*, 2004). My findings add weight to the view that alcohol misuse by teenagers and delinquency are not simply two manifestations of the same problem when violence is involved. The implications of this would include the necessity for providing services specific to each.

So there is then the interesting question as to whether there is a common underlying risk factor for addictive behaviour. If so, it would be expected that this would affect use of other substances, including tobacco and illicit drugs. The association between alcohol and violence was reduced significantly after adjusting for cigarette smoking, and to a lesser extent, cannabis use (52% and 12% respectively). That there remained a significant association between alcohol and violence even after controlling for other substances as well as non-violent delinquency indicates that an underlying propensity for risk-taking, addictive or general problem behaviours does not adequately explain the observed association between alcohol and violence in this study.

Association with drug using peers also explained a large proportion of the apparent relationship, indicating that young people who drank and who were violent were significantly more likely to associate with substance using peers. This is understandable in terms of a number of mechanisms. It is possible, for example, that some of the violence occurred because violent provocation may be more likely to occur among peer groups whose members become intoxicated, disinhibited or are in states of withdrawal of other substances, or who use violence in the acquisition of substances or the means to acquire them. Association with drug-using peers does not therefore adequately explain the relationship between alcohol use and violence.

Adjustment for gender, ethnicity, IQ, depression, impulsivity, temper, neighbourhood violent crime and neighbourhood disadvantage also resulted in small changes in the crude relationship. Of the 24 potential confounders 11 had negligible or no effect on the unadjusted association; they were cocaine, solvent and LSD use, family size, family conflict, family structure, supervision by father, supervision by mother, closeness to mother, closeness to father and population density. Many of these had previously been described as associated with delinquency or alcohol use.

Although a fairly comprehensive set of potential confounders were selected a priori on the basis of previous published research, the list was not exhaustive and there are other potential confounders that were not used in this analysis. It is also possible that other unknown and unmeasured factors have contributed towards residual confounding. Indeed, this is possible in any observational study, but is more likely when, as in this study, there is evidence of confounding observed using the measures available, than when there is little or no evidence of confounding.

Very few previous studies, however, have controlled for as comprehensive a set of risk factors as in the present study, possibly with the exception of the Christchurch Health and Development study (Fergusson and Horwood, 2000), although this study did not control for neighbourhood violent crime or deprivation. In particular, with some exceptions (see Table 1 and Table 2) few studies have controlled for variables that have among those with the greatest effect, namely delinquency and peer substance use.

Adjustment for variables on the Causal Pathway

Although a comprehensive set of confounders was selected a priori based on previous published research, which were adjusted for in the analyses, it is possible that bias may have been introduced because one or more of the variables adjusted for in the analysis are on the causal pathway between alcohol and violence. Such adjustment, known as over adjustment bias (Schisterman *et al.*, 2009) could affect either the estimate or the precision of the estimate of the association between exposure and outcome; specifically, adjustment for variables on the causal pathway has the tendency to reduce the estimate towards zero.

It could be considered that delinquency is the most variable on the causal pathway between alcohol and violence, and therefore adjusting for it may have introduced bias, and reduced the estimate of the effect size. To investigate the extent to which this may have occurred, a repeat of the analyses of the main random-effects model showing the estimated effects of quantity of alcohol, cigarette smoking and cannabis use on violence (Table 38) was carried out with and without any adjustment for delinquency. In the model in which delinquency was removed, there was an increase in the estimates, but the difference was very small. As shown in the model in which delinquency was adjusted for (Table 38), drinking 1-4 drinks or 5 or more drinks is associated with odds ratios of 1.36 (95% CI 1.18-1.58) and 2.14 (95% CI 1.80 – 2.54) respectively. When these analyses are carried out without adjustment for delinquency the estimates for 1-4 drinks and 5 or more drinks are 1.42 (95% CI 1.27-1.60) and 2.26 (95% CI 1.95-2.54). It therefore appears unlikely that adjusting for delinquency has introduced significant bias.

CAUSALITY

As explained in Section II, Chapter 7 the Bradford-Hill criteria(Bradford-Hill, 1965) can be used to assess the evidence that an observed association may be causal.

Temporality

In a causal relationship the exposure must precede the outcome. In this study, the measurement of exposure (alcohol use) preceded the observation of violence by virtue of the prospective longitudinal design and the use of statistical methods appropriate to the design. Given that questions relating to the exposure and

outcome were ascertained repeatedly over 4 waves, there are multiple opportunities to assess the temporal relationship within individuals. As discussed above (see under reverse causality), there remains a difficulty in ascertaining the temporal sequence of alcohol and violence when both arise between waves of data collection.

Strength of association

Analysis of the entire cohort showed that those who regularly consumed 1-4 alcoholic drinks had and an increased odds of violence of 1.36 (95%CI 1.2-1.6%). A 36% increase in the risk of violence may not be considered to be very strong, however among those who regularly drank 5 or more drinks on each occasion the odds of violence was 2.4 (95% CI 1.8-2.5. Whether an odds ratio is categorised as small, medium or large is arbitrary, but a method has been described to categorise odds ratios in an equivalent way to Cohen's d effect sizes which are classified as small (0.2), medium (0.5) or large (0.8). Using the method described by Chen (Chen *et al.*, 2010) odds ratios of 1.7, 3.5 and 6.7 are estimated to be equivalent to Cohen's d effect sizes of small, medium and large respectively when the disease rate is 1% in the non-exposed group. Using this as an approximation (the outcome rate in the non-exposed group in the present study is around 3% as opposed to 1% in the Chen study), the observed association between early onset of drinking and later violence is small.

Dose-response relationship

Evidence of a dose-response relationship is considered to be among the strongest means of providing evidence of a causal relationship. In this study evidence of a dose-response relationship was investigated by analysis of a linear trend between number of drinks consumed and violence, and also be investigating the effect in categories of alcohol consumption (no alcohol, 1-4 drinks, 5 or more). In analyses of both the initiation of violence among those who were not violent at wave I, as well as violence in the entire cohort, after adjusting for confounding, there was evidence to support a dose-response relationship between alcohol consumption and violence.

Although there was no evidence of linear relationship between frequency of drinking and initiation of violence, there is evidence that infrequent alcohol consumption (once a month or less) is associated with violence, but more frequent

alcohol consumption was not. This may be due to the context of the alcohol consumption. For example, if alcohol is consumed regularly within the family home with meals under adult supervision, risk of violence is likely to be very low compared with low frequency but high volume drinking with peers outside of the family home. This is underlined by the finding that there is a linear relationship between both the frequency of binge drinking and frequency of intoxication and violence.

Consistency

A causal interpretation is strengthened when the association is consistently found after multiple replications. In my study, this was the case both in the group who were non-violent at baseline, as well as in the entire cohort. It was also found across several different measures of violence, including measures of all serious violence, fighting, and group violence.

Biological Plausibility

A clear rational or theoretical basis for a reported relationship would provide further evidence in favour of a causal link. Laboratory studies have shown an increase in the likelihood of aggressive behaviour among those intoxicated with alcohol. Although violence is considered to be a complex, multifaceted behaviour, there is an acknowledged lack of carefully conducted research into the complex interactions between alcohol intoxication and aggression(Oscar-Berman and Marinković, 2007). There is clear evidence that alcohol has effects on the brain, which can induce changes in emotional states. The almost universal effect of alcohol inducing mild euphoria at smaller doses, and increasing levels of sedation and impaired coordination at greater doses can be understood in terms of biological mechanisms of the inhibition of specific neurological pathways. The effect of acute alcohol ingestion on an individual depends on the concentration of alcohol in the blood (blood alcohol concentration, BAC). For a given quantity ingested, the BAC varies significantly between individuals, depending on such factors as gender and ethnicity. This in turn varies substantially due to genetic variation, differences in rates of absorption, body mass, and rates of metabolism. The effect of alcohol on the brain varies not only as a function of the BAC, but also on the individual's tolerance to the effects of acute intoxication, their expectancy of

the effect, and the behaviour of others. In general, low doses, of alcohol tend to have a stimulating effect, whereas higher doses cause a depressant effect.

Acute intoxication with alcohol disrupts a number of neurological and cognitive processes, including behavioural inhibition(Marczinski *et al.*, 2005, Marczinski and Fillmore, 2005), and psychomotor performance (Ridderinkhof *et al.*, 2002), and verbal processing(Marinkovic *et al.*, 2004).

The legal BAC for driving in UK, US, and many other countries is 0.08%, however impairment in mental functions can be detected at very low levels of BAC – such as 0.01-0.02%(Koelega, 1995). In simulated driving tests, individuals with BAC less than 0.2% show increased distractibility and poorer attention(Wester *et al.*, 2010).

It has also been shown that following ingestion of alcohol there is a disproportionate impairment in executive functioning tasks including working memory, planning and behavioural control(Peterson *et al.*, 1990) in comparison to other cognitive abilities. Although there is a wide range in individual effects depending on an individual's alcohol tolerance, mild euphoria and relaxation is experienced at BAC of around 0.03-0.06. Unconsciousness may be expected at BAC >0.3, and death at BAC >0.5.

The acute behavioural effects of alcohol are thought to be caused by several processes, however the exact mechanism remains uncertain (Alfonso-Loeches and Guerri, 2011). There is evidence however that alcohol acts on specific membrane proteins, including NMDA-glutamate, γ-aminobutyric acidA (GABA-A), glycine, 5-hydroxytryptamine-3 (5-HT_3), and neuronal nicotinic acetylcholine (nACh) receptors. After the initial direct effect of alcohol on cell proteins, a second wave of actions are initiated through the indirect effects of several neurotransmitters and neuropeptides (Alfonso-Loeches and Guerri, 2011). Among the most important of the receptors with relevance to behavioral effects is the NMDA-glutamate receptor which also play a part in alcohol dependence, tolerance and withdrawal. Alcohol has been shown to inhibit this receptor causing, with chronic alcohol use, causing a compensatory increase in the number of receptors. It is thought that this may contribute to the alcohol withdrawal syndrome on cessation of alcohol use (Kumari M *et al.*). GABA-A receptors are also important in relation to the behavioral effects of alcohol GABA-A is the major inhibitory neurotransmitter in the brain, and alcohol transmission in low to moderate concentrations enhances its transmission.

Other targets for alcohol include neuronal nACh(Narahashi *et al.*, 1999), and 5-HT3(Machu and Harris, 1994).

Alcohol may cause violence by increasing impulsivity. Alcohol has been shown in several studies to impair behavioural disinhibition, which may be due either to the stimulant effect of alcohol, or due to impairment of functions of the prefrontal cortex that are responsible for inhibitory control (Marczinski *et al.*, 2005, Oscar-Berman and Marinković, 2007, Peterson *et al.*, 1990).

Measurement of impulsivity is most often operationalized in the "Go/No-Go" task (e.g. (Abroms *et al.*, 2003)), a task in which participants are instructed to respond to a visual "Go" (which may be for example be a shape or letter) target presented on a computer screen as rapidly as possible, but to inhibit responding to a "No-Go" (a different shape or letter) target. The task measures the number of failures to inhibit response when presented with a "No-Go" target. Alcohol at BAC as low as 0.06% reduces inhibitory control (de Wit *et al.*, 2000, Marczinski *et al.*, 2005). At such BAC the speed and accuracy of responding to the "Go" target is not affected, which suggests that alcohol has a selective effect on inhibitory control, rather than a global effect on disrupting psychomotor performance (Field *et al.*, 2010).

Alcohol may also increase the risk of violence directly. A study of 10 male social drinkers was carried out to assess the effect of acute alcohol administration on aggressive responses during aggression paradigm, and whether such changes were related to baseline trait impulsivity, aggression and anger. The study found that alcohol increased aggressive responding in the study paradigm, and that only impulsivity was correlated with change in alcohol-related aggression. As a consequence, the authors concluded that it is impulsivity that mediated the effect of alcohol on aggression (Fulwiler *et al.*, 2005).

Several studies using fMRI have assessed the acute effects of alcohol on the ability to process socio-emotional stimuli using paradigms of assessment of brain activity when presented with pictures of faces displaying different emotions (happy, fearful, disgusted, angry or neutral). Studies have shown that under the influence of alcohol the amygdala shows an attenuated response to viewing fearful faces, but an increase in activity in response to neutral faces, suggesting that the anxiolytic effect of alcohol may in part be due to a reduction in the ability to detect threatening information, or by reducing the reactivity to perceived threat (Gilman *et al.*, 2012, Gilman *et al.*, 2008, Sripada *et al.*, 2011). This line of research points towards the

effect of alcohol in reducing the perception of threat salience, which may be mediated by reduction in the connectivity between the amygdala and orbito-frontal cortex (Gorka *et al.*, 2013).

There is evidence therefore that alcohol may increase the likelihood of misinterpretation of neutral stimuli, which may be therefore be experienced as threatening, and this in turn could lead to an aggressive response in some people. Alcohol is also thought to disrupt the ability to appraise situations fully. Thus if an individual is faced with a hostile situation when intoxicated, the individual will be more likely to focus on the salient hostile cues, rather than the less salient inhibitory cues (such as the negative consequences of aggression), thus leading to a higher probability of aggression, a theory known as the "myotic effect"(Giancola *et al.*, 2010).

Of interest, areas of the brain known to be implicated in the regulation of impulsive aggression have also been shown to be particularly susceptible to damage by prenatal alcohol exposure. The orbito-frontal cortex and the anterior cingulate cortex are involved in the regulation of behaviour following the appraisal of stimuli in terms of predicted rewards and punishment and applying inhibitory mechanisms (Siever, 2008). Structural abnormalities have been shown in the orbitofrontal cortex among people who have borderline personality disorder (in which impulsivity is a core feature) (Hazlett *et al.*, 2005). Functional imaging has also shown differences in regional blood flow or glucose metabolism; Positron Emission Tomography (PET), for example, has revealed reductions in flow in the lateral, medial and orbito-frontal cortex among people with and without a history of impulsive aggression (Goyer *et al.*, 1994, New *et al.*, 2007).

There is evidence that females are more susceptible to the neurological effects of heavy alcohol use. In an fMRI special working memory task, female adolescent binge drinkers performed significantly worse than non-binge drinkers, and showed decreased activity in frontal, temporal and cerebellar regions, while such differences were not found among males (Squeglia *et al.*, 2011). Chronic alcoholism is associated with abnormalities in the processing of emotional facial expressions, especially anger. Alcoholic patients have been shown to have difficulty in recognising angry faces, and electrophysiological recordings have indicated impaired processing in relation to attention and decision making specific to anger as opposed to other emotions(Maurage *et al.*, 2008).

It is plausible that, in some people, alcohol may result in a general reduction in inhibition that may make violence more likely, or may enhance irritability, however the current biological understanding of these mechanisms is still limited, and therefore caution needs to be applied in attributing effects on biological mechanisms (as opposed to social and contextual reasons) to alcohol. Other theories place the social and contextual factors as more prominent in explaining problem behaviours. In the present study however these factors were adjusted for as far as possible with the available data.

EXPLORATION OF VARIATION OF THE EFFECT OF ALCOHOL ON VIOLENCE WITH AGE

AGE OF ONSET OF DRINKING ALCOHOL AND VIOLENCE

There is evidence that the younger the person is when they start drinking alcohol, the higher their risk of subsequent violence and other problem behaviours. For example, a very large cross-sectional survey of nearly 46,000 people in the USA found that 11% of those who had ever drunk alcohol had had a fight while or after drinking. It was found that there was a linear relationship between age of onset of drinking and the proportion of people who had been involved in fighting after drinking; 34% of those who started drinking before the age of 14 had been involved in fighting, while only 3% of those who started drinking after the age of 21 had done so (Hingson *et al.*, 2001). Those who started drinking alcohol earlier were also those most likely to engage in heavy and frequent alcohol consumption. Even after controlling for patterns of alcohol consumption, earlier initiators of drinking were still more likely to engage in alcohol-related fighting. As described in the introduction a survey of 2,650 17-18 year old students in California and Oregon gathered information about reported age of onset of drinking alcohol. Participants were dichotomised into "early" (age 10-12), and "later" (age 13 or more). Those categorised as early drinkers were significantly more likely to report

being violent when drinking than the later drinkers (41% compared with 23%) They were also more likely to drink more frequently and report a higher frequency of intoxication (Gruber *et al.*, 1996).

Why do those who drink alcohol at a younger age have higher rates of violence? It is possible that drinking alcohol in childhood or adolescence represents a symptom of a broader tendency towards delinquency; the younger that delinquency is manifest, the more deviant the individual may be, including their tendency towards violence. The other possibility is that alcohol causes violence, either by a damaging effect on the brain due to early exposure, or that younger brains are more susceptible to the acute effects of alcohol. I will discuss each of these in turn.

Does Alcohol misuse and the propensity for violence have a common cause?

The clinical diagnostic systems (DSM and ICD) recognise discrete and separate disorders such as conduct disorder (in childhood), antisocial personality disorder (in adulthood), and substance misuse disorders. The extent to which these conditions co-occur, has led some authors to conclude that a common "externalising liability" factor underpins them (Krueger *et al.*, 2002, Krueger *et al.*, 2005). Research also suggests that the categorical conceptualisation of presence or absence of "disorders" is flawed and that an externalising liability is more plausible, which lies on a spectrum underpinned by personality traits, which are themselves expressed on a continuum (Krueger *et al.*, 2007). It is possible, therefore, that the early initiation of alcohol use is an expression of a propensity for antisocial behaviours, including violence. In my study however it should be noted that there was a dose-response relationship between alcohol use and violence, which held even after controlling for other delinquent behaviour, which gives weight to there being a causal association between the two.

There is some evidence from genetic studies that aggressive antisocial behaviour and substance use share a common factor. For example analysis of data from 2,700 twins showed support for a model of shared genetic variance between propensity for substance use disorders and elements of behavioural disinhibition and lack of self control (Iacono *et al.*, 1999). There is further evidence to suggest that

aggression and substance misuse are traits within an overarching "externalising spectrum"(Krueger *et al.*, 2007). Gillespie et al (2009) suggested that shared early environmental risk factors may explain the association. For example, exposure to abuse during critical periods of brain development may permanently alter stress responsivity. As discussed in the introduction however, (Are alcohol use and violence two symptoms of the same problem behaviour syndrome?, page 12), there is evidence from statistical modelling that alcohol use and violence cannot be adequately described by a single common factor (White and Labouvie, 1994), and therefore do not appear to be derived exclusively from a the same underlying cause.

Delinquency cannot in itself be considered to be a causal factor; it is a social construct that describes behaviours that "break rules" within society. Individuals who break one type of rule are more likely to break others, which so the association between alcohol misuse and violence at a younger age could be merely semantic. Alcohol use is common and socially acceptable in adulthood, whereas it is not common or socially acceptable in childhood. Drinking alcohol in childhood may therefore be seen as a form of rule-breaking and may be more likely found among those who have a tendency to break other forms of rules, including those around conflict and violence. It is also likely that young individuals who have peers who use substances may belong to gangs or subcultures in which violence is an acceptable and accepted form of conflict resolution and means of establishing hierarchy (Zdun, 2008). The underlying propensity to either a "problem behaviour syndrome" or an "externalising spectrum" is therefore likely to be underpinned by personality characteristics (see below).

Does alcohol exposure in younger people lead to violence due to damage to their brains?

There is no doubt that repeated exposure to alcohol has a detrimental effect on brain, and that the younger the brain during exposure, the greater the effect. Prenatal exposure to alcohol is associated with a range of physical cognitive and behavioural and abnormalities. Alcohol can disrupt brain growth during the third trimester, the period of rapid development of glial cells and of cerebellar development. Animal studies have shown that a single high dose of alcohol to neonatal mice result in neuronal cell death, especially in the caudate nucleus, and

frontal and parietal regions (Dikranian *et al.*, 2005, Ikonomidou *et al.*, 2000). Other animal study findings indicate that alcohol disrupts several specific stages of cell function or gene regulation. For example depending on the timing of alcohol exposure to the developing brain, alcohol can disrupt cell proliferation and cell growth, migration and differentiation. For example alcohol can disrupt neurepthoelial cell proliferation and migration which occurs between 7 and 20 weeks gestation, causing long-term reduction in brain size, and disruption in corpus callosum formation (Liesi, 1997).

Adolescence is a time of major development of the human brain, particularly the prefrontal cortex. The dopaminergic system within the striatum undergoes substantial change during adolescence – for example animal studies have shown that dopamine receptor density increases in early adolescence then decreases again during later adolescence (Teicher *et al.*, 1995). The GABA system also undergoes considerable developmental change during this period with a substantial increase in the number of GABA receptors and GABA activity (Moy *et al.*, 1998). There is relatively late development in the pre-frontal cortex circuits which are important for impulse control and judgement, which may explain adolescents' propensity for risk-taking, sensation seeking and impulsivity (Alfonso-Loeches and Guerri, 2011). The main processes that continues well into young adulthood (around age 25) are myelination of axons and synaptic pruning (elimination of unwanted synapses). The volume of the prefrontal cortex reduces during this period as neuronal circuits are refined and remodelled. These changes are associated with improvement of response inhibition, working memory and attention (Paus, 2005). It is possible therefore that alcohol is particularly damaging during this period of development. A recent study found that boys with an alcohol use disorder had smaller putamen and thalamic volumes than non-drinking boys and furthermore girls with an alcohol use disorder showed the reverse finding in comparison to non-drinking girls (Fein *et al.*, 2013). It is therefore likely that the younger the age of exposure to alcohol (from the prenatal period onwards), the higher the risk of damage to the brain. It is possible therefore that the chronic effects of alcohol exposure to younger brains may disrupt neurological mechanisms, resulting in either enhanced aggressivity, or reduced self-control. Whether or to what extent these effects are responsible for the increase in violence is not known.

EVIDENCE THAT THE EFFECT OF ALCOHOL ON VIOLENCE DECREASES WITH AGE

As shown in Chapter 14, the association between onset of drinking alcohol and onset of violence is greatest among the youngest in the sample, and the effect reduces with increasing age. Predictive marginal effects showed that drinking 1-4 drinks on each occasion was associated with an increased risk of violence during adolescence, but the trajectory then tended to converge with that of non-drinkers. Drinking 5 or more drinks, however, was associated with a greater risk of violence well into adulthood, although the trajectories tended to converge by age 30.

It is possible that younger people are more susceptible to the detrimental effects of alcohol. There is evidence from previous research that alcohol has differential effects on the adolescent compared with the adult brain; for example adolescents have more memory impairment during acute intoxication than adults(Acheson *et al.*, 1998), but are less susceptible (in animal studies though there are no comparable studies in humans) to effects of impairment in motor co-ordination (White *et al.*, 2002a, White *et al.*, 2002b) and sedation(Little *et al.*, 1996). This is important because the sedative and motor-impairing effects of alcohol serve as a limiting factor in human drinking behaviour. If adolescents are less susceptible to the acute negative effects of alcohol than adults, it is possible that they may consume more alcohol, and achieve higher blood alcohol concentrations that adults.

There is evidence, however, that adolescents are less susceptible to the effects of alcohol on the GABA system; alcohol activates GABA receptors and chronic exposure to alcohol results in a reduction in the number of GABA receptors. Upon cessation of alcohol, there is insufficient GABA activity, which can result in seizures. One animal study found that adolescent rats who had had 5 consecutive days of alcohol administration followed by a substance to induce seizures experienced seizures at a similar rate to adult rats similarly treated. However the seizures lasted longer in the adult rats suggesting that alcohol had less effect on the GABA system in adolescent as opposed to adult rats(Acheson *et al.*, 1999).

Three other studies as mentioned in the introduction have found that the association between alcohol and violence is present only during younger cohorts. In the study by Huang (Huang *et al.*, 2001) out of several cross-lagged associations tested, only alcohol use at age 16 and aggression at age 18 were significantly associated. The authors concluded that there may be a unique effect of alcohol use

on mid-adolescence leading to aggression. The study by Wells (Wells *et al.*, 2004) found that the severity of alcohol problems was associated with violence at age 16-21 but not age 21-25. And the study by Scholes-Balog (Scholes-Balog *et al.*, 2013) similarly found that the relationship held between age 13 to 15, but not age 15 to 17.

It is of course possible that the decrease in the association between alcohol and violence with increasing age is due to changes in the strength of confounders over time. For example, it has been shown that deviant peer relationships are associated with violence by the individual, but the effect is only present on younger people (14-15 years), and not older ones (20-21)(Fergusson *et al.*, 2002).

In my study it was not possible to correct for all time-dynamic confounders and it is therefore possible that changes in the strength of the relationship over time can be explained by changes in the confounders; I was only able to adjusted for these as though they were static.

Overall, however, and taking into consideration evidence from previous studies that have indicated a possible greater effect in younger people than older ones, there appears convergent evidence of a reduction in the strength of the effect of alcohol on violence with age.

PATTERNS OF DRINKING AND VIOLENCE

There was a linear trend between quantity of alcohol consumed on each occasion and violence. Frequency of alcohol consumption was not associated with violence after adjusting for the amount consumed on each occasion. It is likely, therefore, that the severity of intoxication induced by binge-drinking is implicated in the association with violence. As with the association between alcohol and violence discussed above for binge-drinking and violence, two possible explanations are that both binge-drinking and violence may represent a common propensity for problem behaviours, or there may be direct influences of alcohol on behaviour making violence more likely. Drinking large quantities of alcohol per drinking occasion ("heavy episodic drinking" or "binge-drinking") has previously been shown to be associated with alcohol-related aggression (Dukarm *et al.*, 1996, Swahn and Donovan, 2004, 2005).

Analysis of data from the 1970 British Birth Cohort Study(Viner and Taylor, 2007) showed that binge drinking at age 16 was associated with a variety of adverse outcomes by age 30, including homelessness, illicit drug use, accidents, fewer qualifications, school exclusions and psychiatric morbidity. The study was only able to adjust for a limited number of potential confounders, including socioeconomic status of father, maternal education status at age 16 and own social class at age 30, and baseline level of outcome under study where available. This study showed that binge drinking, but not habitual frequent drinking was associated with these adverse outcomes.

The same birth cohort provided information about patterns of binge drinking in adolescence and later adverse outcomes, including criminal offending, though not specifically violence (Viner and Taylor, 2007). Over 11,000 people participated. Nearly 18% of people reported binge drinking at age 16. When followed-up at age 30, those who had reported binge drinking were more likely than those who did not to have a wide range of adverse outcomes; they were more likely to have alcohol dependence(OR 1.6) have left school without qualifications (OR 1.3) having been expelled (OR 3.9), have used illicit drugs in the last 12 months (OR 1.7), be a heavy smoker (OR 1.7), have a history of homelessness (OR 1.6), and have had a criminal conviction (OR 2.2). The authors then tested whether the effects of binge drinking were different from a pattern of frequent habitual drinking. They found that regular habitual drinking was also associated with a greater likelihood of alcohol dependency, but was not associated with other adverse outcomes such as criminal offending and school exclusions, and in fact was associated with higher socio-economic status.

Another survey from the UK, called the 1998/99 Youth Lifestyle Survey 9YLS), (Richardson and Budd, 1993) surveyed 4,848 12-30 year olds, and was designed to measure self-reported offending. A subgroup of 1,336 18-24 year olds formed the basis of an analysis of the relationship between been drinking and offending (Richardson and Budd, 2003). The authors used two definitions of binge drinking. Firstly they defined it for male participants as drinking 8 or more units and for female participants 6 or more units in a single day. In a second definition, they characterised binge drinking as getting drunk at least once a month. They reported this to be a better measure as a definition based on units takes no account of individual's weight, alcohol tolerance or gender, which may affect the influence that alcohol has on the individual. Based on the preferred second definition, they

classified 39% of the sample as binge drinkers. Binge drinkers were over 4 times more likely to report that they had been involved in a violent crime in the previous 12 months compared with non-binge drinking regular drinkers, and more than 8 times more likely to report violence than non-drinkers. Frequency of drunkenness was strongly associated with overall offending even after controlling for confounders. Drug use, by contrast, was more predictive of theft than violence. Binge drinkers were also more likely to report fighting after drinking than other regular drinkers or non-drinkers. Age was also found to be an important factor; 18-20 year olds were twice as likely to report getting into a physical fight than 20-24 year olds.

The above studies indicate that binge-drinking is associated with numerous adverse outcomes and problem behaviours. There are several studies that have shown that patterns of repeated exposure to high concentrations of alcohol followed by withdrawal as in binge drinking are directly harmful to the brain. A study showed that rats administered with alcohol three times a day over 4 days to simulate binge drinking had neurodegeneration of the corticolimbic circuit, and showed poorer responding on special learning tasks (Obernier *et al.*, 2002). Studies of binge-drinking adolescents have found that the adverse effects on the brain may be chronic (Crews *et al.*, 2000). One study found that students who had a history of binge drinking performed worse on memory tasks after consuming alcohol than students without a history of binge drinking (Weissenborn and Duka, 2003). Consistent with this finding, hippocampal volume has been shown to be smaller among those who abused alcohol during adolescence (De Bellis *et al.*, 2000). Hippocampal volume was also found to be correlated with age of onset of alcohol abuse. Interestingly however, there were no differences in overall cerebral volume, cortical grey or white matter volume, corpus callosum volume or amygdaloid volume, suggesting a specific effect on the hippocampus. The hippocampus is thought to have a modulating role in aggression and structural abnormalities have been found in people who are antisocial and aggressive (Critchley *et al.*, 2000, Raine *et al.*, 2004), and therefore may provide a direction for further studies on the neurobiological mechanisms of alcohol related violence.

It is not possible from cross-sectional studies to ascertain whether the observed abnormalities were present before the onset of alcohol abuse, but three longitudinal studies have investigated the effect of alcohol consumption on groups of young individuals with no past history of heavy alcohol consumption. Girls and

boys aged 12-14 were prospectively studied over a 3-year follow-up period. Girls who began either moderate or heavy drinking during the follow-up period showed a greater deterioration in neurocognitive tasks, particularly tasks involving visuo-spatial memory. For boys, a greater deterioration in attention was found compared with those who did not initiate heavy or moderate drinking (Squeglia *et al.*, 2009). There is evidence too that that there are differences at baseline among those who go on to drink heavily; a prospective study found lower parietal and frontal activation detected by fMRI during a visual working memory task, suggesting that different neural response patterns predict later alcohol misuse (Squeglia *et al.*, 2012). Similarly, a prospective study over 9-months among first year university students showed that among those that when on to binge drink had significantly slower cerebral activity as observed by greater latencies of ERPs in response to auditory stimuli in comparison to those that did not drink whereas there had been no difference between the groups at baseline(Maurage *et al.*, 2009).

The association between binge-drinking and violence therefore appears to be multidirectional. It is possible that pre-existing neuropsychological differences exist among those who binge drink, and these differences may also predispose to violence. It is also likely that both the acute and chronic effects of heavy drinking are causally related to violence.

NUMBER NEEDED TO PREVENT

The number needed to prevent (NNP) is the number of individuals it would be necessary to protect form exposure to a risk factor to prevent one occurrence of the outcome. I found that it would be necessary to stop 54 adolescents (median age 15) from drinking alcohol to prevent at least one incident of serious violence within the next year (95% CI 23–671). No published data were found from other studies that reported a NNH with respect to alcohol and violence. Although 54 may appear to be a large number of individuals who would have to be prevented from drinking, to put this into context, it is of the order accepted as worthwhile in public health interventions; for example, the number of healthy individuals who would have to be given influenza vaccinations to prevent one case of influenza has been estimated to be 71 (95% CI 76-128) (Demicheli *et al.*, 2014). Further, the calculated NNP for alcohol to prevent violence is far lower than the estimated number of people who would have to be stopped from using cannabis to prevent one case of schizophrenia

in the following year; among the highest risk age group this NNP is estimated to be 2,800 (95% CI 2,018-4,530) and 10,870 (95% CI 6,786-22,732) among the lower risk age group (Hickman *et al.*, 2009).

The NNP for binge drinking was 47 (95 % CI 17-189). This means that somewhere between 17 and 189 adolescents who engaged in binge drinking would need to reduce their drinking to prevent one acting violently within the next year.

The analysis suggests that intervention to reduce or prevent binge drinking may be effective in reducing violence. The interventions that have been employed are varied and have had variable success. In the traditional "public health" model of prevention of alcohol problems (Blane, 1976) there are three tiers of prevention. They are primary – interventions to prevent or reduce the incidence of new cases of violence, thus focused on education to change attitudes and behaviours towards drinking alcohol; secondary – to reduce the seriousness, frequency or duration of violence, thus projects aimed at early identification, treatment and resolution of alcohol problems; tertiary – to reduce the longer-term disabilities and disadvantages associated with violence, such as loss of friends and loved ones, employment and, generally, social status and of freedom (imprisonment), longer term programmes to sustain abstinence or low levels of drinking and limit relapse. A systematic review of the effectiveness of psychosocial and educational interventions for the primary prevention of alcohol misuse in young people identified 56 studies, of which 41 were randomised controlled trials and the remainder were non-randomised but had a control group (Foxcroft *et al.*, 2003). The review found that in 20 of the 56 studies there was no evidence of effectiveness. There was heterogeneity in the types of interventions and in the target groups, but one intervention that stood out as potentially valuable was an intervention called the Strengthening Families Program (SFP). This was an intervention delivered to 293 families in the USA in which families with children age 11-13 attended 7 weekly sessions lasting 2 hours each in which parents and children were taught skills to communicate better, appropriate discipline, and managing strong emotions. (Spoth *et al.*, 2001). The intervention was thus quite labour intensive and time-consuming which would likely limit the scalability of the intervention.

The other main preventive public health measure is to make alcohol less available. Legislation technically protects younger people from accessing alcohol unless

under supervision, although there is little evidence that it is much enforced, at least in England and Wales. Legislation has been used to restrict access to alcohol altogether, for example in the 1920s in the USA. Minimum pricing of alcohol by unit, and/or increasing taxation on alcohol is another route, as well as limiting the premises or hours during which it may be available. There have been arguments in favour of banning advertising of alcohol and sponsorship of events by alcohol merchandisers (Alcohol Health Alliance UK, 2013), but there is little hard evidence to support any of these routes, and paradoxical effects of interventions may occur, such as the rise of organised crime during prohibition in the USA.

Much previous research has implicated alcohol in violent behaviour, and data from my study suggests that young people may be most vulnerable to becoming violent after alcohol consumption. While most previous studies have focussed on relationships within a time period or indirect proximity to the violence, I chose to consider the impact of underage drinking on young teenagers who had not previously been violent. Thus, my addition to knowledge in this field is that if non-violent teenagers with a median age of 15 (range 13-17) start to consume alcohol, they double their risk of becoming quite seriously violent in the following year, and the risk increases considerably if they are binge drinkers. A number of anticipated potential confounders do affect that relationship, but it does remain through multi-variable analyses and the fact that it is dose related adds weight to the probability that it is the alcohol *per se* which is having an effect. Although the risk of violence seems to fall off over time, estimates of the 'numbers needed to treat' suggest that the target figures would be well within accepted public health ranges, and much harm might be prevented. If it were possible to prevent just 54 young people from binge drinking at least one serious violent incident should be prevented. There have already been programmes directed at achieving reduction of drinking by young people, and review suggests only modest success (Foxcroft and Tsertsvadze, 2012) , although, consistent with the extent of confounding influences confirmed in my study (and others), there is promise in the suggestion that programmes should engage in a much wider range of tasks than simple alcohol educational and advisory services.

I also considered mechanisms by which alcohol may be exerting this effect on onset of violence. Accepting that there is unlikely to be one single explanation, the fact that the adolescent brain is at a particularly critical stage of development and potentially exceptionally vulnerable to toxins, the combination of my findings of a

dose relationship with onset of violence and a changing relationship over time does point to cerebral mediation. Future research should try to tease out such effects because they would have relevance to the nature and extent of violence prevention. If the relevant damage is primarily effected in the early teenage years, then that renders prevention programmes imperative; if the relevant damage may occur at any time, and may be cumulative, then prevention of early teenage drinking alone may merely delay the problem. The fact that later onset of drinking seemed to have less impact on onset of violence would tend to add weight to the argument for early intervention.

CHAPTER 17 CIGARETTE SMOKING, CANNABIS USE AND VIOLENCE

SUMMARY OF FINDINGS

Self-reported cigarette smoking among adolescents who were not already violent at a mean age of approximately 15 was associated with about twice the likelihood of self-reported violence within the next as non-smokers. The association was present in both crude and unadjusted models, however the strength of the association was reduced slightly in the adjusted models. When the period of follow-up was extended to include any violence over the 13 years of the study, there was an association with violence among those who smoked 60 cigarettes or more in the preceding month in the unadjusted analyses, but there was no association between cigarette smoking and violence once other variables were adjusted for. Among the entire cohort, taking into account changes in smoking and other substance use, as well as adjusting for static risk factors, there was an association with onset of violence among heavier (more than 60 cigarettes in a month) but not lighter smokers. The strength of the association was greatest among the younger smokers and decreased with age. When males and females were analysed separately, there was a small association between smoking and violence among the males between the age of 14 and 19; there was no association among females.

There was no association between cannabis use and incident violence in either crude or adjusted analysis. However among the entire cohort, there was an association with violence. Both lighter cannabis users (up to 10 times in a month) and heavier (over 10) had an association with violence of a similar strength. The effect was greatest among the younger participants and decreased as they got older. The effect was similar in males and females, however among males even in their mid-20s, cannabis was estimated to have a small association with violence, but in females, the association was present only until their late teenage years.

There are several possible explanations for the observations, and although many of the issues are similar to those discussed in Chapter 16, I will discuss them here again where they apply to the association between cigarette smoking, cannabis use and violence to aid interpretation.

EXPLORATION OF CAUSAL INFERENCE

REVERSE CAUSALITY

Among the cohort who were not already violent, smokers were more likely to report subsequent violence than non-smokers. As described in Chapter 16, minor aggression is developmentally normal, and precedes substance use. For example, a prospective longitudinal study in Iceland of 14-year-old non-smokers found that antisocial behaviour was significantly associated with subsequent daily smoking at age 17 after controlling for confounders. (Adalbjarnardottir and Rafnsson, 2002). Although antisocial behaviour rather than aggression was measured in this study, it is possible that those who are predispositionally aggressive are more likely to smoke cigarettes. However serious violence, the focus of this study is rare and would not be considered to be within developmentally normal limits. The analysis of incident violence in the present study was aimed at reducing the possibility of reverse causality, however it is possible that some individuals who were violent at baseline did not report their violence and were incorrectly included in the analyses.

The analysis of the entire cohort using random effects models incorporated both levels of self-reported smoking and violence at each wave and *change* in individuals levels of smoking and violence would also help disentangle the temporal relationship. It is possible that between waves of data collection there may have been initiation of both smoking and violence, or discontinuation of both smoking and violence but the exact temporal sequence of change in both behaviours could not be determined within the design if the study. The extent to which this may have occurred cannot be known.

BIAS

MISSING DATA AND CASES LOST TO FOLLOW-UP

There was very little missing data for the questions regarding cigarette smoking (less than 1% missing) at any of the waves of data collection. With regards to cannabis use there was missing data for less than 2% of participants in waves I and II, and less than 0.4% missing at wave III and IV. Very little bias could therefore be introduced by differential absence of data between those who were violent and those who were not.

As stated in Chapter 16, the response rate after wave I ranged from 77% to 89%. The analyses of incident violence (Chapter 12) made use of sample weights, which corrected for the probability of retention in the sample and therefore mitigated against possible bias from study attrition. It was not possible however to use sample weights in the random-effects models as this capability was not available within the software used for analysis. Loss to follow-up could have resulted in an over-estimate of the association between either cigarette smoking or cannabis use and violence only if substance users who became violent were less likely than non-substance users who became violent to drop out, or if non-substance users who did not become violent were more likely to drop out. However, it is more likely that substance users who were violent were more likely to drop out due to other chaotic or problem behaviours, and therefore it is possible that the observed relationships in the random effects models may be an under estimate.

INFORMATION BIAS

Reporting and Recall Bias

As stated in Chapter 16, bias could be introduced if there was a systematic difference in inaccurate responses between comparison groups. As stated, all of the measures were based on self-report. It is possible that among those who were violent there was a tendency to exaggerate their cigarettes smoking or cannabis use, or that those who smoked or used cannabis exaggerated their violence. There is no evidence overall that participants in Add Health responded differently about their smoking habits as compared with participants in other surveys. By comparison, among 9th grade students (age 14-15) from a different nationally representative survey in 1997 (the same year that participants of a similar age were surveyed in Add Health, 33% reported that they had smoked at least one cigarette in the 30 days prior to the survey(Centers for Disease Control and Prevention, 1998), compares with 34% who responded positively to the same question at wave II (grades 7-11) in the present study.

With regards to cannabis use, approximately 13% of respondents at wave I reported they had used cannabis at least once in the 30 days prior to the interview. The prevalence is slightly lower than that recorded in a similar survey from the same year (1995) among 9th grade children in USA in which 20% reported they

had used cannabis in the past 30 days(Kann *et al.*, 1996). It is not known whether respondents may have under estimated their cannabis use relative to this survey, whether participants may have over-estimated their cannabis use in the comparator study, or whether there were true differences. For bias to be introduced, there would need to be a systematic difference between reports of cannabis use among those who became violent compared with those who did not. It those who became violent tended to exaggerate their cannabis use, or those that were not violent minimised their use, the real association between cannabis use and violence would be expected to be even lower.

Given that the question relating to cannabis use required participants to recall whether they had used cannabis only over the previous month, it is possible but unlikely that the results are susceptible to significant recall bias.

Misclassification Bias

Bias could be introduced if either the exposure status or outcome status of individuals has been systematically misclassified. There would appear to be only a small likelihood of misclassification of smoking or cannabis use as all participants completed the computerised survey and were given questions in the same format and wording and their responses were recorded electronically and therefore required no further interpretation.

CONFOUNDING

As stated in the methods in Chapter 9, the association between either cigarette smoking, or cannabis use and violence was adjusted for a comprehensive set of potential confounders. The variables were chosen from among those that had been shown in previous studies to be associated primarily with alcohol and violence. Similar risk factors exist for both alcohol use and other substance use, and therefore the same set of confounders was used in all analyses. Although a fairly comprehensive set of potential confounders was selected *a priori* on the basis of previous published research, the list was not exhaustive and there are other potential confounders that were not used in this analysis. A thorough literature review of the predictive factors for illicit drug use among young people (Frisher *et al.*, 2007) revealed several factors that were not analysed in the present study, which included, other life events, Attention Deficit Hyperactivity Disorder and other mental health problems, religion, participation in sport and drug availability.

Of these, perhaps the greatest omission from the list is the failure to include a measure for adverse life events as a potential confounder. It is possible that adverse life events (including being the victim of violence) may increase the propensity to use substances and to be violent. Adverse life-events were found in the Christchurch Health and Development Study to be associated with violent offending(Fergusson and Horwood, 2000) and with the initiation of drug use(Fergusson *et al.*, 2008). It is possible therefore that there was a higher proportion of individuals who experienced an adverse life event in the group who initiated both alcohol misuse and violence than those who did not which may have explained part of the findings and resulted in an over-estimate of the association between substance use and violence. It is also possible that other unknown and unmeasured factors have contributed towards residual confounding. Indeed, this is possible in any observational study, but is more likely when, as in this study, there is evidence of confounding observed using the measures available, than when there is little or no evidence of confounding.

CAUSALITY

As explained in Section II, Chapter 7 the Bradford-Hill criteria(Bradford-Hill, 1965) can be used to assess the evidence that an observed association may be causal.

Temporality

The design of the study was longitudinal and analyses were carried out to detect which exposures preceded the outcome. Although as stated earlier it is possible in the random effects and GEE models that between violence preceded cigarette smoking or cannabis use between waves, however there would be no way of detecting this from the available data. In the analysis of incident violence however the exposures clearly preceded the outcome.

Strength of association

In the early teenage years (mean 15 years), there was twice the risk of onset of violence in the year after onset of smoking. Whether this is considered a strong association is a matter of opinion. The advice of (Chen *et al.*, 2010) is that it is a small effect. Nevertheless, if this were considered to be a causal relationship, an exposure that doubles the rate of violence among adolescents could be considered to be important.

Early smoking was not associated with incident violence when the length of follow-up was extended to include any violence over the entire study. This would be expected, as there is no evidence that smoking in adolescence would have a causative effect on violence many years later. Among the entire cohort, when changes in smoking were modelled and therefore more proximal effects of exposure on outcome could be modelled, there was a small (OR 1.3-1.8) but potentially important association with violence.

There was no association between cannabis use and incident violence. However, among the entire cohort, over the duration of the study, and taking into account changes in cannabis use and in analysis that could model effects over adjacent waves, there was a small association with violence (OR approximately 1.5).

To put the size of the odds ratio into context with those found in other areas of medicine and public health, a review of the decision making of expert committees which applied a version of the Bradford-Hill criteria to review causal inference between smoking and cancer, causal association was not claimed for associations with an odds ratio of less than 3.0 (Parascandola *et al.*, 2006). Indeed it was the strength of the association, (along with dose-response and biological plausibility) that carried the most weight for the expert committees in drawing conclusions as to causality. However causal associations are accepted in other area with far smaller effects sizes, and which influence public health policy (Public Health England, 2014).

Dose-response relationship

With regard to cigarette smoking and incident violence, there was no evidence of a dose-response relationship. Among the entire cohort, there was no association between light smoking (up to 60 cigarettes in a month) and violence, but there was an association among heavier smokers (those who smoked more than 60 cigarettes a month). If there is a causal relationship between smoking and violence, it is possible that the mechanism may involve an increase in irritability caused by nicotine withdrawal (see below). A dose-response effect may therefore not be expected to be linear. Very heavy smokers may not experience periods of nicotine withdrawal in the same way that more moderate smokers may do. Moderate smokers would more likely have frequent periods of relative nicotine withdrawal compared to heavy smokers, and therefore may be at greater risk of irritability leading to violence. This hypothesis however needs further clarification and

conclusions can not be drawn from the present study. There was no dose- response relationship between cannabis use and violence.

Consistency

Only one previous longitudinal study was identified that considered the relationship between cigarettes smoking and violence; this showed no relationship. Very few longitudinal studies have reported on the relationship between cannabis and violence and the resulting evidence is mixed. My study was designed to fill the lack of current knowledge in this area, so estimates of consistency are not, by definition, possible, although a systematic review and meta-analysis of the associations between cannabis use and aggression (Derzon and Lipsey, 1999) also showed that the relationship was strongest in the younger age groups compared with the older age groups.

Biological Plausibility

In the introduction I touched on evidence from animal laboratory studies that nicotine reduces irritability and aggression (e.g. (Johnson *et al.*, 2003), however the biological mechanisms are not known. Several studies have found that prenatal exposure to cigarette smoking is associated with a higher rate of subsequent violent offending. For example, a twofold increase in violent offending by age 26 was found in the offspring of mothers who smoked during pregnancy compared with those whose mothers did not smoke(Rasanen *et al.*, 1999). Two of the constituents of cigarette smoke -carbon monoxide and nicotine - have been shown to be neurotoxic (Olds, 1997). Prenatal exposure to cigarette smoke disrupts the development of noradrenergic neurotransmitter system, and may contribute to the brain deficits found in adult offenders (Raine, 2002). It is possible that exposure to tobacco smoke during childhood and adolescence may also affect brain development and maturation which may increase the likelihood of violence.

There is evidence that nicotine withdrawal increases irritability and aggression among smokers (Cherek *et al.*, 1991, Parrott and Zeichner, 2001). The levels of nicotine in the blood rise rapidly during cigarette smoking and are at the peak at the end of smoking a cigarette. The blood levels then decline rapidly over the next 20 minutes as nicotine is absorbed and metabolised. The distribution half-life averages approximately 8 minutes (Benowitz *et al.*, 2009). The possible mechanism

that links smoking to violence may therefore be due to frequent periods of relative nicotine withdrawal among smokers inducing greater irritability which may lead, in a small proportion of times to aggression and violence.

The mechanism however is likely to be still more complex, and may be linked to an interaction between one or more of the constituents of cigarettes with the neurobiological underpinnings of personality traits. One study categorised participants as either high or low trait hostility. Positron Emission Tomography (PET) of participants showed that those with low trait hostility showed no change in brain metabolism when administered nicotine, but high hostility participants (both smokers and non-smokers) showed a dramatic change in brain metabolism throughout virtually all cortical and sub-cortical areas bilaterally(Fallon *et al.*, 2004). In a double-blind randomised controlled trial of non-smokers who were given a 2mg dose of inhaled nicotine during a stress-inducing task found that among women, the nicotine reduced their ratings of aggression, whereas in was enhanced it in males.(File *et al.*, 2001). It is therefore possible that nicotine has a differential effect on people according to gender or personality characteristics, which may be associated with violence, however further studies are required.

Coherence

There is evidence that cigarette smoking exacerbates stress; in once study the daily mood pattern of smokers showed normal moods during smoking (rather than increased relaxation), and worsening moods, tension and irritability between cigarettes reflecting nicotine depletion(Parrott, 1999). A study (as mentioned in the introduction) which shows a reduction in aggressive responses when nicotine was administered (Cherek, 1981) was carried out among smokers who had been smoking for at least 4 years.

The findings on the question of a relationship between cannabis and violence are mixed; some studies have found a high rate of violence among cannabis users, some a lower one, and others have found no effect (Ostrowsky, 2011). Part of the discrepancy may be accounted for by the fact that some studies take a measure of any cannabis use, some heavy use and some dependency. There are several hypotheses that may explain any relationship between cannabis and violence. Cannabis use may induce a different emotional state, including paranoia, fear or panic, which may lead to violence (Moore and Stuart, 2005), or depersonalisation (the feeling that one is disconnected from one's body, or observing one's body

from outside), which could be frightening and increase the likelihood of violence (Moore and Stuart, 2005). There is however little empirical evidence to support this. There is also evidence that cannabis use is associated with psychosis (Zammit *et al.*, 2002, Zammit *et al.*, 2008), and there is a well established association between psychosis and violence (Taylor, 2008).

As with the hypothesis that nicotine withdrawal may increase violence, cannabis withdrawal has also been hypothesised to increase the risk of violence (Moore and Stuart, 2005, Ostrowsky, 2011). Several studies have shown that cannabis withdrawal increases irritability, anger and aggression (Budney and Hughes, 2006, Hoaken and Stewart, 2003, Kouri *et al.*, 1999, Milin *et al.*, 2008). It is therefore possible that cannabis withdrawal, rather than cannabis intoxication may be associated with violence.

Although there are possible mechanisms to support a causal association, it is also possible that the apparent association in this study is due to unmeasured or "residual" confounding. Although the analyses were adjusted for the effect of a large and fairly comprehensive set of potential confounders, there remains the possibility that there were others important confounders, such as social position. This interpretation of residual confounding to explain the apparent relationship between cannabis and violence, and tobacco and violence also has implications on the interpretation of the relationship between alcohol and violence. If it is accepted there is residual confounding, there is also likely to be residual confounding in the relationship between alcohol and violence, and therefore these findings may have been over estimated. The extent to which this may have occurred can not be known.

One approach that could be used to test the causal association between exposures and outcome is Mendelian Randomisation (Davey Smith and Ebrahim, 2003, Sheehan *et al.*, 2008). The method is used to test the causal association of an exposure on an outcome when confounding is likely, but not fully understood. The method involves the identification of a variable, usually a genetic variant (known as an instrument) that is reliably related to the exposure, but independent of the confounders, and of the outcome (once the exposure and confounders are accounted for). The ALDH2 allele has been used as an instrument associated with alcohol intake to test the casual association between alcohol and blood pressure (Chen *et al.*, 2008). Genetic variants associated with heaviness of tobacco use

(rs1051730) have also been identified and used as instruments in Mendelian Randomisation studies to investigate causality between tobacco use and depression and anxiety (Gage *et al.*, 2013). This method could potentially be used in the future to investigate the causal association between tobacco use and violence if this allele was measured in the entire sample.

Investigation of the extent to which the Big-5 personality factors, according to the self-rated Mini-IPIP, were associated with violence, and whether alcohol mediated this showed that agreeableness was inversely associated with violence in both men and women. Here, reference was to all variables measured in wave IV only, so focus shifted from concern about onset of violence to the association between alcohol, personality and violence. Alcohol mediated approximately 11% of the effect in males, but there was no evidence of an effect in females. Similarly, anger-hostility was strongly associated with violence in both sexes, but alcohol mediated the effect only in males (approximately 20% of the total effect). Extraversion was associated with both violence and alcohol use in males and females. Alcohol accounted for 15% of the effect of extraversion on violence in males and 29% in females.

An alternative to the theory that alcohol and violence stem from the same underlying cause (the common cause theory) is that the relationship between alcohol use and aggression is modified by personality factors. This is the theory known as the "conditional/interactive" theory (Pernanen, 1981). There is evidence that alcohol consumption is more likely to result in aggression in those with higher dispositional aggression (Bailey and Taylor, 1991). In a laboratory setting, intoxicated participants demonstrated higher levels of aggression in the form of delivery of electric shocks in a competitive task against a simulated participant, a version of the Taylor Aggression Paradigm (Taylor, 1967) than sober participants. When intoxicated, those who reported higher levels of dispositional aggression at baseline were significantly more likely to deliver shocks without provocation, and shocks of higher intensity than when sober, or compared with intoxicated participants of low dispositional aggression. In another study using this paradigm, Those with higher levels of dispositional aggression were was found to predict high levels of aggression in intoxicated but not sober participants under conditions of low provocation(Miller *et al.*, 2009)

Other studies have found that individuals with higher levels of dispositional anger were more likely to exhibit alcohol-related aggression. In further laboratory tests using a modified version of the Taylor Aggression Paradigm, dispositional anger

was positively related to aggression in all subjects, but alcohol increased aggression especially in those with higher dispositional anger (Giancola, 2002a, Parrott and Zeichner, 2002). Similar findings were observed with regards to irritability (Giancola, 2002b), and so called "hostile ruminations" (the tendency to ruminate on feelings and intentions associated with seeking revenge and retaliation for perceived provocation (Borders and Giancola, 2011). Perceived provocation was the factor that most strongly elicited violence in all subjects (Giancola, 2002a, Giancola *et al.*, 2002). A further study using similar methods found that dispositional anger *per se* was not associated with intoxicated aggression except in those with poor anger control, suggesting that the degree of self-reported self-control was the crucial factor (Parrott and Giancola, 2004).

With regard to the 5-factor model of personality, a meta-analysis of 15 studies with a combined number of over 4,500 participants, examined the relationships between personality characteristics and antisocial behaviour and found strong inverse relationships with agreeableness (weighted mean effect size -0.41) and conscientiousness (weighted mean effect size -0.25). There was a positive relationships with neuroticism (weighted mean effect of 0.12), but no significant relationship with extraversion or openness(Miller and Lynam, 2001).

My findings were, therefore consistent with these previous findings, in that traits which I expected to be associated with violence were so. At this stage, without allowing for alcohol consumption, agreeableness was significantly inversely associated with violence among men. Women are generally found to score higher on measures of agreeableness than men, but previous studies have not investigated males and females separately for its relationship with violence. A new finding from my study is that the relationship between agreeableness and violence is similar in both men and women. There are, however, differences in the extent to which alcohol mediates this relationship. In men, alcohol accounted for around 14% of the relationship between agreeableness and violence, whereas in women, there was no evidence of an effect of alcohol. This implies that the relationship between personality, alcohol and violence may be different in men and women.

A surprising finding in this study was that there was no direct relationship between neuroticism and violence. However, I found a significant association between angry-hostility (a facet of neuroticism within the five-factor model) and violence, in both men and in women. Although there appeared to be a larger effect of angry-

hostility on violence in women than men, there was no evidence of alcohol mediating this effect in women. In contrast, in men there was evidence that a significant part of the effect (approximately 19% of the total effect of angry-hostility on violence) was mediated by alcohol.

Although previous studies have shown that neuroticism is associated with violence, this finding suggests that angry-hostility may be its component in this relationship. With the exception of impulsivity, the other facets of neuroticism (anxiety, self-consciousness, depression and vulnerability), are not generally considered to be contributory factors to violence. This suggests that the facets which are clustered within each personality factor may not act equally, or indeed even in the same direction in causing behaviours, and therefore a more detailed understanding of relationships between personality, violence and alcohol may be achieved by investigating relationships at the level of the facet in addition to the core personality factors.

With regard to the other personality factors, both extraversion and openness were associated with violence. Previous findings as to the relationship between extraversion and aggression are mixed. Jung described the extravert as someone who had a tendency to action rather than thought, and there is some face validity to the notion that such individuals may be more likely to act aggressively. Eysenck believed that extraversion was higher in offenders than non-offenders (Egan, 2009), however he revised this view when impulsivity and extraversion were considered to be independent of one another (impulsivity is considered to be a facet of neuroticism in current five factor models of personality (Costa and McCrae, 1992). Although some studies have shown an association between extraversion and aggression, for example in males who engage in intimate partner violence(Fechter and Snell, 2002), the majority of studies to date have shown no relationship with aggression (e.g.(Sharp and Desai, 2001). In my study, alcohol accounted for a substantial part of the relationship between extraversion and violence in both men and women, and this may explain variation in results in studies which do not control for the effect of alcohol. Extraversion has been consistently shown to be associated with alcohol use and misuse in general population samples (Cooper *et al.*, 2000, Malouff *et al.*, 2007, Peterson and Morey, 2005) (Ruiz *et al.*, 2003, Stewart *et al.*, 2001, Vollrath and Torgersen, 2002), although not in samples drawn from those with alcohol use disorder or psychiatric diagnoses (Malouff *et al.*, 2007, Martin and Sher, 1994, Trull and Sher, 1994). It is likely that extraverted

individuals are more likely to socialise, and in western societies, are more likely to do so in places where alcohol is available and where exposure to situations where violence might be precipitated.

In my study, openness was associated with violence in men, but not women, and it was associated with alcohol in both sexes. A previous study found a positive relationship between a measure of physical aggression and openness, but not between self-reports of violent behaviour and openness (Barlett and Anderson, 2012). Other studies have shown no relationship between openness and aggression (Barlett and Anderson, 2012, Gleason *et al.*, 2004, Sharp and Desai, 2001), or with alcohol use or alcohol use disorders (Ibanez *et al.*, 2010, Lackner *et al.*, 2013, Malouff *et al.*, 2007, Ruiz *et al.*, 2003). Differences may have arisen due to variation in the measures of aggression and violence, and whether the effect of alcohol was controlled. I focused on serious violence as the outcome and found relationship between being open in these terms and being violent. It is not immediately apparent why individuals who report higher levels of imagination and interest in abstract ideas are also more likely to report violence, and further studies are required to further investigate this.

A negative relationship between conscientiousness and alcohol use was found. This is consistent with previous studies (Ibanez *et al.*, 2010, Kashdan *et al.*, 2005, Malouff *et al.*, 2007, Martin and Sher, 1994, Trull and Sher, 1994). Previous studies have variously found either a negatively association with aggression(Sharp and Desai, 2001, Tremblay and Ewart, 2005) or no relationship with aggression (Barlett and Anderson, 2012). I found a small association with violence, but 40% of the total effect was mediated by alcohol, again highlighting the importance of taking both personality traits and alcohol into account when trying to understand how either relates to violence.

There were some limitations. Firstly, the measure of personality traits was a brief questionnaire, each trait measured by only 4 items. Nevertheless, this instrument has been shown to be a valid and reliable measure of the Big-Five personality traits(Donnellan *et al.*, 2006), and has been demonstrated to have acceptable psychometric properties as applied to data in the current study (Baldasaro *et al.*, 2013). In addition, my confirmatory factor analyses showed adequate model fit for the 5-factor personality structure using these items. Secondly, all measures were based on self-report. Validity might have been improved by the availability of

information from multiple sources. Audio-computer assisted self interview (ACASI) on laptop computers was, however, used for sensitive health and health risk behaviour questions in all waves. This technology has been found to improve the quality of self-report of sensitive information (Turner *et al.*, 1998a). Thirdly, although the effect sizes give an indication of the direction and relative magnitudes of the effects of personality traits on violence, their magnitudes are difficult to translate to clinical meaning when the units are in standard deviations and the measures are factor scores. Fourthly, although the model showed adequate statistical fit of the data, alternative models are possible that may fit the data equally well or better. My primary model was guided by theory and provided the best model fit. We tested an alternative model which was theoretically possible which did not fit the data as well, but others are possible which were not tested as they did not fit with our hypotheses or previous theory. In addition, for this part of the study, the data were cross-sectional, and therefore the direction of association as specified in our models provides the best statistical fit of the data, but does not really allow inferences about direction of effects. Furthermore, there may be confounding of the relationships. I controlled for the effect of age, and analysed separately by gender but other confounders for which I did not control may be present. Indeed the model indicated that only around 20% of the variance in violence were explained by the personality factors and alcohol. This is the first study however to investigate the relationship between personality factors, alcohol and violence in a community setting.

LIMITATIONS

The main limitation is that the analyses are cross-sectional. As personality was only measured at wave IV, analyses were confined to data from this wave only Although personality tends to be fairly stable over time, it maybe influenced and shaped by environmental factors such as substance misuse or treatment (de Groot *et al.*, 2003). It was therefore considered not to be a stable time in-variant characteristic for the purpose of longitudinal analysis.

The limitation therefore of cross-sectional analysis is that the direction of association between the variables can not be determined. Thus it is possible that the final model, although providing the best statistical "fit" compared with the alternatives that were tested, is inaccurate and the direction of associations maybe

opposite or bi-directional. Further research is therefore needed to test the hypothesised model in other samples.

Despite this significant limitation with regards to the investigation of causation, one of the strengths of SEM is in developing new hypotheses with regards to the relationships between variables to inform new research where there is little established knowledge. To my knowledge, there are no previously published models as to the relationships between personality subtypes, alcohol and violence, and therefore despite the very clear limitation of the cross-sectional nature of these analysis, this preliminary model could be used as a basis for further research as to the inter-relationships between personality, alcohol and violence in other samples.

CHAPTER 19 CONCLUSIONS AND FUTURE RESEARCH

Violence is a serious event which may have multiple adverse consequences for victims, perpetrator and society, and there is a need to understand more about aetiological mechanisms to aid prevention and intervention. The principal aims of my study were to investigate the relationship between alcohol use and the risk of onset of violence during adolescence and early adulthood in the general population. Secondary aims included investigation of as the relationships between frequency or quantity of alcohol use and risk of violence, and to investigate whether the relationship between alcohol use and violence varied with age.

There was strong evidence of an association between early heavy alcohol use and onset of violence. This was a dose dependent relationship. There was strong evidence that the amount of alcohol consumed rather than frequency of alcohol consumption was associated with an increased risk of violence. There was also evidence that cannabis use and cigarette smoking were associated with an increase in risk of onset violence. These relationships were not dose related. Like alcohol, their effects were most marked at younger ages. It is unlikely that the introduction of bias, either in the selection of participants or in differential loss to follow up adequately explains these associations. There was substantial confounding of the association, however there remained strong evidence of an association following adjustment for a range of measured confounders such as delinquency and other substance use. After taking previously published findings into account, the implications of my findings suggest that alcohol does have a casual contribution to onset of violence, although the effect is smaller than may have been expected from cross-sectional association studies. This effect was greatest in early adolescence and diminished with age.

It is possible that in this study the frequency of the data collection, and having only 4 opportunities for data ascertainment did not provide sufficient resolution to detect proximal effects of alcohol on violence, but the large size of the sample, which was representative of the US general population, the variation in age within the cohort, the variation in time between data collection points, the long follow up period, good study retention rate, and the use of comparable measure at each time point allowed for comprehensive modelling of the association. As the perpetration of serious violence however is fortunately quite a rare event in adults, more

frequent data collection at the expense of sample size would be unlikely to have provided more insight.

Although there was evidence of an association, the effect size was small. Nevertheless, assuming the origins of violence are multifactorial, the identification and reduction of each modifiable cause could help to reduce the overall burden of violence in communities, and reduce the effects on individuals. This study would suggest that efforts towards primary prevention of or reduction in violence by reducing heavy alcohol use could have a useful impact and be best focussed on adolescents rather than adults. Assuming that a successful intervention could be implemented, around 40 adolescents would have to be prevented from binge drinking in order to prevent 1 from perpetrating serious violence within the next year, but that is well within the range of preventive calculations considered worthwhile by public health workers. A combination of primary preventative public health measures could be implemented, including education, limitation of access to alcohol, reducing social, family and neighbourhood factors that may contribute to heavy drinking. A lack of empirical data for efficacy of alcohol use reduction programmes with teenagers means that further developments will be needed in this area.

Once drinking and violence have become established, then secondary and tertiary prevention strategies become more important in order to limit further damage, The mechanism of the effect of alcohol on violence is complex and there is evidence that the effect of alcohol on violence varies with personality types. Low agreeableness is particularly associated with violence and there is evidence that alcohol medicates this relationship in men but not in women. My findings suggest that such intervention strategies must take account of personality traits as well as alcohol consumption among men – particularly traits of disagreeableness, angry-hostility, openness and extraversion. Among women, there seems to be much less interaction between alcohol consumption, personality traits and violence, so this perhaps more complex approach to intervening may be less justified.

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