INTRODUCTION

The term ‘adolescent’ is an adjective describing a young person in the process of developing from a child into an adult and dates from the late 18th century (Oxford English Dictionary). It is derived from the Latin verb ‘adolescere’ which means ‘to grow up’. This short paper will review patterns of drinking in adolescence and the risk factors that are thought to predispose to the development of alcohol use and other co-morbid disorders in this age group.

Alcohol is the world’s third largest risk factor for disease and contributes to 4% of the global burden of disease (Rehm et al., 2009). It is estimated that ~2.5 million deaths each year are directly attributable to alcohol, with 9% of deaths in the 15- to 29-year age group being alcohol-related (WHO, 2011).

When data from the World Health Organization’s Global Burden of Disease study were used to calculate cause-specific disability-adjusted life years (DALYs) for young people aged 10–24 years, the main risk factors were found to be alcohol (7% of DALYs), unsafe sex (4%), iron deficiency (3%), lack of contraception (2%) and illicit drug use (2%) (Gore et al., 2011). The contribution of other risk factors to disease, such as tobacco use, low physical activity and overweight/obesity only emerged in mid-to-late adulthood. These findings suggest that public health strategies should focus on child and adolescent health, and that adolescent drinking should be given priority (Gore et al., 2011).

Regular alcohol use, binge drinking and other risk-taking behaviours such as smoking, substance use and risky sexual behaviour emerge in adolescence and there is evidence that these behaviours tend to cluster together (Wiefferink et al., 2013). The adolescent brain, especially the hippocampus, may be particularly vulnerable to the effects of alcohol (Welch et al., 2013), thus predisposing the young drinker to alcohol, mental health and neuro-cognitive problems which can persist into adulthood (Hanson et al., 2011; Welch et al., 2013). Young people who start to drink before the age of 15 years are reported to be four times more likely to meet criteria for alcohol dependence at some point in their lives (Grant and Dawson, 1997). Early alcohol use is associated not only with more regular and higher levels of alcohol use and dependence in adulthood, but also with more mental health and social harms (McCambridge et al., 2011).
consumption had decreased since 2001 when the figure was 26%. Mean alcohol consumption in 2011 was 10.7 units and the median 7 units. Drinking was associated with smoking and drug use, and having truanted from school.

A recent study of English students aged 13–14 years and 15–16 years found that most had had an alcoholic drink (70 and 89%, respectively), and that the first drink had most often been taken at about the age of 12–13 years, and usually in the company of an adult on a special occasion (Bremner et al., 2011). One fifth of 13–14 years old who drank were drinking weekly, and the figure was 39% for the 15–16 year olds. One quarter of the older students had consumed six or more drinks on the last occasion they had taken a drink. The 13–14 year olds were more likely to have been drinking alcopops in the 7 days before the survey, whereas the 15–16 year olds were most likely to have been drinking beer, lager, spirits or liqueurs. By the age of 15–16 years, 79% of the students had been drunk and two-thirds of this group (66%) said they drank to get drunk at least once a month.

The Australian Secondary Students’ Alcohol and Drug Survey (ASSAD) is carried out every 3 years. The 2011 survey, the tenth in the series, included responses from just under 25,000 secondary students (White and Bariola, 2012). About three out of four students had tried alcohol at some point in their lives, 51% consuming alcohol in the 12 months before the survey. Just under one-fifth (17%) had consumed alcohol in the 7 days before the survey, 8% of 13 year olds and 37% of 17 year olds. About one-fifth (19%) of 17 year olds had consumed more than four drinks on at least one of the preceding 7 days. The proportion of students drinking in 2011 was less than that found in the 2008 and 2005 surveys.

**COHORT STUDIES**

What are the consequences in adulthood of late adolescent drinking? McCambridge et al. (2011) carried out the first systematic review of general population cohort studies where data on baseline alcohol consumption had been obtained from adolescents between the ages of 15 and 19 years, and follow-up data had been obtained from the same cohort at time points that were at least 3 years apart. The review included 54 studies, of which 35 were multiple reports from ten cohorts including 9 reports from the (all male) Swedish Conscript Study (SCS). Almost one half of studies (n = 26) were from the USA with the remainder coming from Sweden, Britain, New Zealand, Australia, Finland and the Netherlands (McCambridge et al., 2011). The main conclusion was that late adolescent alcohol consumption persisted into adulthood and was associated with alcohol problems, including dependence. Non-alcohol outcomes such as the mental health and social consequences of adolescent drinking could not be fully explored due to lack of evidence. The SCS evaluated the risk of premature death with late adolescent drinking after 15, 20 and 25 years and found that by the age of 34 years heavier drinkers were twice as likely as moderate drinkers to have died (Andreasson et al., 1988, 1991; Romelsjo et al., 1999). This finding was attenuated by the age of 39, but the main causes of death at both time points were car crashes (mainly at younger ages) and suicide (at older ages) (Andreasson et al., 1991). Good psychosocial adjustment did not protect heavier drinkers from an increased risk of premature mortality (Andreasson et al., 1991).

A recent paper from the SCS series reported that alcohol use in adolescence, particularly ‘risky’ use, was associated with an increased risk of obtaining a future disability pension (Sidorchuk et al., 2012). The association was stronger for early disability, indicating that risky adolescent alcohol use is a risk factor for diminished work capacity.

McCambridge et al. (2011) noted that few studies had addressed family influences and only one had investigated genetic aspects (Viken et al., 2007). The Viken et al. (2007) study of Finnish twins suggested that genetic and environmental influences on the development of alcohol problems between the ages of 18 and 25 years were different for men and women, such that genetic influences remained stable over time for men, but declined for women.

A recent study from a prospective UK birth cohort reported on alcohol use at 10, 13 and 15 years and found that by the age of 15 over half of the boys and girls had consumed alcohol and one-fifth reported drinking in a binge fashion (MacArthur et al., 2012). There were no gender differences in drinking behaviour. Higher alcohol consumption at 15 was associated with a significantly higher prevalence of engagement in other risk behaviours at 16 years, in particular substance use and sexual risk behaviours. Those who met criteria for hazardous drinking at 16 were six times more likely to engage in substance use behaviours than those who did not meet these criteria.

Another study of the same birth cohort used longitudinal latent class analysis to categorize alcohol use in the 13–15 year olds as low, medium and high, in terms of frequency and quantity of alcohol consumption (Heron et al., 2013). When they were 16, the young people completed a postal alcohol use disorders identification test (AUDIT) questionnaire: 29% met criteria for hazardous alcohol use (AUDIT score >8–15) and 5.6% met criteria for harmful use (AUDIT score >16). Being in the high class for either drinking frequency or consumption was associated with an 8- to 10-fold increase in odds of harmful alcohol use at 16 years.

A systematic review of longitudinal studies that examined the association between childhood socio-economic status and alcohol use later in life found that there was little evidence to support the association (Wiles et al., 2007).

Alcohol use, and particularly binge drinking, is associated with sleep disorders in a dose–response relationship (Popoviv and French, 2013). These findings were reported from the National Longitudinal Study of Adolescent Health which examined a sample of just over 14,000 adolescents and young adults. Further work is needed to explore the association.

There is clearly a need for high-quality long-term prospective cohort studies to investigate the long-term consequences of adolescent drinking. There is already enough evidence in the literature to warrant interventions to reduce drinking in adolescents (McCambridge et al., 2011).

**RISK FACTORS FOR DRINKING IN ADOLESCENTS**

The adolescent brain undergoes profound neuro-developmental change, in turn influenced by genetic, environmental and sex hormonal factors (Arain et al., 2013). Glutamatergic neurotransmission is predominant and the maturation of neural circuits facilitates social-emotional development (Nelson et al., 2013).
At the same time puberty manifests itself in outward bodily change. The risk factors for adolescent AUDs can be divided into environmental, genetic and phenotypic. Genetic-environmental interaction determines individual alcohol use and AUDs.

Environmental factors
As adolescents become more autonomous so the influence of the peer group becomes more important and family influences wane (Bremner et al., 2011). Adolescence sees a clustering of risk-taking behaviours such as smoking, drinking, drug-taking and sexual activity. Peer effects on risk-taking are strong in this age group, and adolescents affiliated with substance-using peers are at greater risk of engaging in similar behaviours themselves (Gardner and Steinberg, 2005). Peer acceptance is a potent social reward for adolescents (Rubin et al., 2006; Guyer et al., 2012) and is associated with high self-esteem and social competence. Having friends who drink increases the likelihood that young people will drink too. Young people are also influenced by how much their friends are drinking. Having older friends and spending more time with drinking friends are likely to promote excessive drinking (Bremner et al., 2011).

Parental expectation and involvement in social activities has been shown to moderate alcohol use (Nash et al., 2005; Wichers et al., 2013) and religious affiliation also shows a protective effect. Factors that influence drinking, drinking frequently and drinking to excess include lower levels of parental supervision, exposure to a close family member who drinks or becomes intoxicated, easy access to alcohol and positive expectations of alcohol (Bremner et al., 2011).

Genetic factors
Genetic predisposition accounts for about half of the risk in the development of alcohol dependence. Adolescents with a positive family history of alcohol problems are at greater risk of developing an alcohol problem, and at a younger age, than their peers with negative family histories. Genetic factors may have more influence on drinking behaviour in late than in earlier adolescence (Rose et al., 2001).

Certain childhood characteristics are thought to increase the risk of adolescent AUDs and early identification of these characteristics can be helpful in preventing or attenuating the risk (Thatcher and Clark, 2008). For instance childhood psychological dysregulation is a behavioural phenotype that reflects an individual’s vulnerability to developing an AUD in adolescence (Tarter et al., 2003; Thatcher and Clark, 2008). Other characteristics that have been identified and can be measured, but cannot be seen, are known as endophenotypes. Endophenotypes are not an element of the disorder but are associated with it, contribute to individual vulnerability and are seen in the families of affected individuals (Laucht et al., 2007). A range of endophenotypes has been identified as markers for AUDs in young people, including behavioural sensitivity to alcohol and event-related potentials (e.g. P300). Schuckit and colleagues have shown that a low level of response (LR) to alcohol is associated with heavier drinking and alcohol problems (Schuckit et al., 2005, 2008). Adolescent children of alcoholics have been shown to have an abnormal P300 response and abnormalities in brain structure and function (Hill and Steinhauer, 1993; Iacono et al., 2002; Yoon et al., 2006).

Sensation seeking and behavioural disinhibition are associated with an increased risk of developing substance use disorders in adolescence and may mediate genetic risk (Laucht et al., 2007; Iacono et al., 2008).

Externalizing problems in childhood, in particular conduct disorder, have been shown to predict adolescent alcohol and substance use disorders (White et al., 2001; King et al., 2004; Fergusson et al., 2007; Young et al., 2008). A longitudinal study from the West of Scotland (N = 2586 pupils) explored the causal effects of alcohol (mis)use and antisocial behaviour in pupils followed up between the ages of 11 and 15 years (Young et al., 2008) and the findings suggested that antisocial behaviour was the main predictor of alcohol misuse and alcohol-related trouble in this under-age cohort. An American study of 429 rural youths found that delinquency at the age of 11 was a positive predictor of alcohol use at 16 for both boys and girls (Mason et al., 2007). However other studies have reported a reciprocal relationship (D’Amico et al., 2008) and Iacono et al. (2008) propose that a common genetic liability to behavioural disinhibition underlies the co-occurrence of early onset substance use disorders and these other disorders. A recent study of male twins found that genetic risk of externalizing disorder and peer deviance predicted the greatest risks of unfavourable alcohol trajectories (Wichers et al., 2013).

Adolescents with attention deficit hyperactivity disorder appear to have an increased risk of drug use disorders but the evidence for an association with AUDs is mixed (Molina and Pelham, 2003; Molina et al., 2007, 2012). Parental knowledge may confer a protective effect in relation to alcohol use in adolescents with ADHD (Walther et al., 2012).

Of internalizing disorders, only depression at the age of 11 years was shown to have a significant relationship with substance use at the age of 14 years (King et al., 2004).

Adolescents with poor affect regulation and depression or who are experiencing high levels of environmental stress may drink or use drugs to self-medicate as a maladaptive coping mechanism. The association between low mood and alcohol use appears to be stronger in adolescents with fewer conduct problems (Hussong et al., 2008).

Adolescents are able to tolerate higher levels of alcohol than adults and they are also more likely to experience the positive effects of alcohol. This may contribute to the development of binge drinking.

INTERVENTIONS
A meta-analysis of treatments for adolescent substance abuse found that treatment was effective in reducing alcohol use and that individual interventions performed better overall than family interventions (Tripodi et al., 2010). The number of studies included was relatively small (n = 16 and 26 outcomes), so the results should be interpreted with caution. However both individual and family-based behavioural treatments were effective in promoting long-term reduction in alcohol consumption (i.e. at 12-month follow-up). Behavioural interventions, either of an individual or of a familial nature, appear to be associated with long-term change. Further work is needed to identify the most effective interventions (Tripodi et al., 2010). Caselanos-Ryan et al. (2013) argue for the development
and testing of preventative interventions to target early key risk factors for substance use disorders. Such interventions should be embedded in a public health policy that seeks to minimize or delay early onset of alcohol use in adolescents.

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REFERENCES


