ALCOHOL USE IN ADOLESCENCE
A longitudinal study of predictors of early and excessive drinking and their association with important life outcomes

Frøydis Enstad
Department of Child Health and Development
Division of Physical and Mental Health
Norwegian Institute of Public Health

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Summary

In this thesis, predictors of early onset of drinking and excessive drinking, and the prospective relationship between early and excessive drinking and important life outcomes in young adulthood, such as hazardous drinking and educational and work-related outcomes, were examined. Data were drawn from three population-based longitudinal studies, two from the Norwegian cultural context and one from Victoria, Australia. All three surveys have collected data in several waves following children from early childhood, through adolescence and into young adulthood. The data used in the three studies presented here were analysed using logistic regression analysis (Paper 1), modified Poisson regression analysis (Paper 2) and latent growth curve analyses by using a cohort sequential design (Paper 3).

In the first study (Paper 1), we found that antecedents of early onset of drinking (EOD) and intoxication (EOI) differed. All the included predictor variables (e.g., smoking, conduct problems, deviant friends) describing the adolescents’ own and friends’ norm-braking behaviours were significantly related to EOI, but not to EOD. This suggests that norm-braking behaviours may be particularly important in understanding the development of EOI. In the second study (Paper 2) we found, contrary to our expectations, that although early onset of excessive drinking (EOE) was a marginally stronger predictor, both EOD and EOE increased the risk of hazardous drinking in late adolescence/young adulthood. The pattern was similar across Norwegian and Australian samples. Furthermore, this pattern remained after comprehensive control for potential confounders, indicating that early adolescent drinking, regardless of level, is an indicator of alcohol-related problems in late adolescence/young adulthood. In the third study (Paper 3), we found that alcohol intoxication (AI) in early adolescence was related to lower educational attainment and risk of detrimental labour market outcomes in young adulthood. The relationship was, however, confounded by familial and individual risk factors, suggesting that early AI is a marker for risk rather than a specific risk factor per se. Interestingly, AI in late adolescence and young adulthood (> 20 years) showed either no significant relationship to educational and labour market outcomes, or was even related to more positive outcomes, such as higher income and less risk of disability. The positive associations remained after adjusting for covariates.

Overall, the results suggest that although antecedents differ, early drinking behaviours, regardless of level, indicate risk of later alcohol-related problems, a risk that persists at least to late adolescence/young adulthood and functions as a marker of risk of adult disadvantage in
work and education. While early AI functioned as a marker of risk of detrimental young adult outcomes, AI in young adulthood, however, indicated no such risk. The findings suggest that preventing young people from drinking in early adolescence may have some impact on their drinking patterns in late adolescence/young adulthood. Furthermore, the findings demonstrate the importance of addressing multiple individual, familial and peer risk factors in early adolescence, not only in prevention of young adult drinking patterns, but also as an effort to prevent detrimental outcomes in other important adult life outcomes, such as education, income and risk of unemployment and disability.
List of papers

Paper 1:

Paper 2:

Paper 3:
Alcohol intoxication across adolescence and educational and labour market outcomes in adulthood: A population-based longitudinal study. *Submitted to Alcohol and Alcoholism*. 
**Abbreviations**

Alcohol use:
AI: Alcohol intoxication
AUDIT: Alcohol Use Disorders Identification Test
EOD: Early onset of drinking (without having been intoxicated or drinking excessively)
EOE: Early onset of excessive drinking
EOI: Early onset of intoxication

Others:
AUS: Australia/Australian
CI: Confidence interval
IYDS: International youth development study
NOR: Norway/Norwegian
OR: Odds ratios
PBT: Problem Behaviour Theory
PR: Prevalence ratios
TOPP: Tracing Opportunities and Problems study
YiN: Young in Norway study
1. Introduction

Most people do not develop long-lasting problems associated with alcohol use, and for many, alcohol use has mostly positive connotations. However, alcohol is also associated with a wide range of health problems (e.g., cirrhosis and traffic injuries) and social problems (e.g., relationship problems and violence) (Babor, 2010). Studies show that alcohol use is responsible for 3.8% of all global deaths and 4.6% of global disability-adjusted life-years are attributable to alcohol use (Rehm et al., 2009). In high- and middle-income countries, the costs associated with alcohol-related health problems and social harms amount to more than 1% of the gross national product (Rehm et al., 2009). Finding an effective method to prevent the negative consequences of alcohol use has thus been of great interest and importance both in research and social policy.

Most of those who drink have their onset of drinking in adolescence (Chen & Kandel, 1995). Adolescence is furthermore considered a vulnerable developmental period, as many physiological, psychological and social changes take place during this period of life (Dahl, 2004). Thus, the adolescent years have been a primary focus in research on alcohol use and alcohol use prevention for several decades. Still, drinking among adolescents continues to be a major health and social concern, representing a burden to the individual and to society (Babor, 2010). In research on adolescent alcohol use, the concern has revolved particularly around the issue of identifying factors predicting early onset of drinking and the relationship between early onset, subsequent alcohol use, and important life outcomes in adulthood (for reviews, see Donovan, 2004; Maimaris & McCambridge, 2014; Marshall, 2014; McCambridge, McAlaney, & Rowe, 2011; Zucker, Donovan, Masten, Mattson, & Moss, 2008).

The concern with early onset of drinking has its roots in a series of studies that consistently demonstrate strong association between early onset and subsequent high levels of alcohol consumption (e.g., Pitkänen, Lyyra, & Pulkkinen, 2005), alcohol-related problems (e.g., Hingson, Heeren, Jamanka, & Howland, 2000) and alcohol disorders (e.g., DeWit, Adlaf, Offord, & Ogborne, 2000; Grant & Dawson, 1997). Based on these and other studies demonstrating similar associations, it has been suggested that postponing the age of onset would prevent subsequent alcohol-related problems. The simplicity of this idea that “the earlier you start drinking, the worse you end up,” has made such studies highly influential in shaping alcohol prevention programmes in many Western countries, including Norway. However, in the last couple of decades this position has been questioned, and critical reviews
of the literature have concluded that the empirical evidence for causal inference is weak (Rossow, 2006). Moreover, many of the studies in the field suffer from limitations, such as retrospective designs and lack of control for possible confounding factors (Kuntsche, Rossow, Engels, & Kuntsche, 2016). As a result, there are still questions to be answered related to predictors of early drinking behaviours and the role of early onset for subsequent drinking patterns and important life outcomes.

One important question that needs to be addressed is whether early onset of more than a few sips of alcohol is predictive of detrimental life outcomes or whether only the consumption of more excessive amounts of alcohol in early age is related to such risk. Longitudinal studies have suggested that greater attention should be paid to the relationship between early excessive drinking and later problems, as such drinking seems to play a more important role for negative development than drinking smaller amounts of alcohol (Kuntsche et al., 2013; Warner & White, 2003). Still, few have contrasted early drinking of smaller amounts and early excessive drinking to different outcomes later in life.

A somewhat related question is whether excessive drinking at later developmental stages has just as negative effects as early excessive drinking. Longitudinal studies of adolescent excessive drinking have generally established that such behaviour is a developmental phenomenon that typically emerges between the ages 14 to 18, peaks in the late adolescence/young adulthood period, and subsequently declines (Chassin, Pitts, & Prost, 2002; Johnston, O’Malley, Bachman, & Schulenberg, 2012). However, few have examined whether excessive drinking in early adolescence leads to different health and psychosocial outcomes in early adulthood than excessive drinking at later developmental stages (for a review, see McCambridge et al., 2011). Fewer still have examined age-specific aspects of excessive drinking in relation to educational and work-related outcomes. More high-quality longitudinal studies with a long follow-up period has therefore been called for (Marshall, 2014; McCambridge et al., 2011).

The main objective of this thesis is to contribute to a better understanding of some of the key aspects of adolescent drinking: (a) predictors of early onset of drinking and excessive drinking; (b) the prospective relationships between early onset of drinking and excessive drinking and important life outcomes in young adulthood, such as hazardous drinking, education, income and risk of unemployment and disability; and (c) whether the association between excessive drinking and later life outcomes changes across adolescence and young adulthood. Research in this thesis is mainly guided by an empirical approach and uses three prospective longitudinal studies to address the above-mentioned topics.
The following sections in the introduction provide an overview of definitions and operationalisations used in the scientific literature of early adolescent drinking behaviours and how they are used in the current thesis, and a description of alcohol use in a cultural context, with a particular focus on Norway. The last section provides an account of the historical theoretical backdrop that has provided the framework for the understanding of risk and protective factors in the field of adolescent alcohol use, and furthermore reviews some of the suggested mechanisms explaining the link between early onset and detrimental adult outcomes. Findings from empirical studies and recent reviews are presented in relation to these frameworks and some critical knowledge gaps are identified. These knowledge gaps have guided the choice of overall aim and specific research questions in the current thesis, which are described in chapter 2. Chapter 3 describes the methods while the results are summarized in chapter 4. In the final chapter (chapter 5) the results are discussed, rounded up by implications of the findings and a concluding remark.

1.1 Early onset of drinking

The terms age at first drink, early onset and early initiation are often used interchangeably in the scientific literature (for a review, see Kuntsche et al., 2016) although the concepts have different connotations. Age at first drink refers to the age at which one first used alcohol, while the concepts of early onset or initiation refers to alcohol use beginning prior to a certain age, with a cut-point at some age considered early. All three concepts however, originate form the idea that the earlier one begins, the worse, as described above. However, what can be considered early, is not always clear cut. One could imagine that any drinking occurring before the legal drinking age, could be considered early. The legal drinking age, however, differs across countries, from 16 years in for example the Netherlands to 21 years in the US. Despite this fact, the age at which most adolescents start to drink is very similar in many Western countries, where around 50% or more of 15 -16 year olds have initiated alcohol use (Kraus & Nociar, 2016). Still, studies have adopted very different cut-offs for early onset, and definitions vary from age 11 (Guttmannova et al., 2012) to age 16 (Moss, Chen, & Yi, 2014). However, most researchers’ working definition is to include drinking behaviour that begins before the age of 14 (Donovan & Molina, 2011; Skidmore, Juhasz, & Zucker, 2011).

Definitions of onset or initiation also vary considerably (for a review, see Kuntsche et al., 2016). Definitions range from having consumed any quantity of alcohol (e.g., Dooley & Praise, 2007; Geels, Vink, Van Beijsterveldt, Bartels, & Boomsma, 2013), via more than just a sip or two of alcohol at least three times during the last year (e.g., Scholes-Balog, Hemphill,
Reid, Patton, & Toumbourou, 2013), to having consumed at least one standard unit of alcohol (i.e. half a bottle of beer, one decilitre of wine or one quarter of a decilitre of spirits) (e.g., Pedersen & Skrondal, 1998). It has also been defined as having at least a few sips of alcohol without parental permission (e.g., Oxford, Harachi, Catalano, & Abbott, 2001). And finally, alcohol initiation has also been defined as having been drunk or admitting to using alcohol occasionally (e.g., Pitkanen, Kokko, Lyyra, & Pulkkinen, 2008). Although the upper limit of what is included in the concept seems to differ widely, it is now generally acknowledged that sipping and tasting should not be considered when determining onset. Sipping and tasting is one of the earliest drinking behaviours that children engage in, but does not seem to reflect psychosocial proneness for problem behaviour (Donovan & Molina, 2008). The most common definition of onset refers to having had “more than a few sips” or having had at least one “drink” (for a review, see Kuntsche et al., 2016). Still, the lack of measurement consistency in the literature on early onset has been criticized, as a wide variety of drinking experiences are treated as the same phenomenon (Kuntsche et al., 2016).

We have in this project attempted to respond to some of this critique. In line with most of the definitions in the literature, early onset of drinking was in this project defined as having had more than just a few sips, thereby excluding sipping and tasting. How the cut-point for early was set, is described in section 1.2 below. When referring to the studies comprising this thesis, the abbreviation EOD will be used to describe this concept (i.e., early onset of drinking without having been intoxicated or drinking excessively, see also section 3.3.1). In reviewing and discussing other empirical studies, the term “early onset” will be used to cover all the different operationalisations used in the literature to describe the onset of drinking (e.g., early age at first drink, early onset of drinking, early initiation, drinking debut).

1.2 Early onset of excessive drinking

Although alcohol use in adolescence, and particularly heavy episodic drinking, has received substantial attention in research on youth, the first, or earliest, experiences of excessive drinking have received relatively little attention. In recent years, however, early onset of intoxication, excessive drinking, or intoxication debut, are increasingly being used as concepts. Still, compared to the number of empirical studies on early onset of drinking more than a few sips, studies of early onset of more excessive drinking as a phenomenon are relatively rare, despite the fact that there seems to be growing evidence that larger effects can be observed in relation to the onset of intoxication than age of first drink (Maimaris & McCambridge, 2014).
In the identified studies on early onset of excessive drinking the concept is based on subjective reports of “feelings of drunkenness” such as “did you feel high or drunk at alcohol initiation” (e.g., Warner & White, 2003), or asking about frequency of drinking so much that the respondent felt clearly intoxicated (e.g., Rossow & Kuntsche, 2013), or in the same manner as questions tapping age at first drink, asking how old respondents were when they first got drunk or felt influenced by alcohol (e.g., Hingson, Heeren, Winter, & Wechsler, 2003; Ystrom, Kendler, & Reichborn-Kjennerud, 2014). Even as young as 14 years of age, adolescents seem to understand well the concept of drunkenness, and their perception of drunkenness has been found to correlate well with their estimated blood alcohol concentration (Lintonen & Rimpelä, 2001).

The concern related to what can be considered early, as described above concerning early onset, also applies to the concept of early onset of excessive drinking. Typically, the onset of excessive drinking occurs somewhat later than the onset of drinking (Hellandsjø Bu, Watten, Foxcroft, Ingebrigtsen, & Relling, 2002). However, the peak period for risk of onset of excessive drinking is in the same range, between 14-16 years (Jackson, 2010). In all the three samples used in the studies comprising this thesis, questions about drinking smaller amounts of alcohol and excessive alcohol use were measured at each wave of data collection. We thus set the cut-point for early as close in time as possible to the age when most adolescents have not yet started to drink or been intoxicated. In practice, this meant drinking smaller amounts or more excessively at age 14.5 or younger (Paper 1), 15 or younger (Paper 2) and 13 or younger (Paper 3).

In this thesis, an attempt was made to nuance the knowledge about the role of early adolescent drinking behaviours by separating “early drinkers” from “early excessive drinkers”. This separation is based partly on an assumption that drinking excessively at an early age may be more consistently considered a social problem across cultures, than having more than just a few sips. Furthermore, as recent research suggests that early excessive drinking may be of greater importance for a wide range of subsequent adverse outcomes than early drinking experiences without intoxication, these concepts may have different antecedents and may also relate differently to adult outcomes. These are some of the topics explored in this thesis.

When referring to the studies comprising this thesis, different abbreviations will be used to describe early excessive drinking (see also section 3.3.1); EOI (early onset of intoxication, Paper 1), EOE (early onset of excessive drinking, Paper 2) and AI (frequency of alcohol intoxication). In reviewing and discussing other empirical studies, the term “early
onset of excessive drinking” will be used to cover the different operationalisations used in the literature (e.g., early age at first intoxication, early onset of intoxication, intoxication debut, feeling drunk at initiation).

1.3 Alcohol use and the cultural context

Countries vary to a large extent when it comes to alcohol policies, drinking culture and norms regulating drinking behaviours. As a consequence, alcohol consumption levels and drinking patterns also vary across countries. Norway has one of Europe’s most restrictive alcohol policies (Österberg & Karlsson, 2003). Historically, the policy is influenced by a strong temperance movement fighting drunkenness and health problems caused by alcohol (Hauge, 1998). Later on, the total consumption model, introduced by Skog (1985), gave arguments to maintain a restrictive policy. Skog demonstrated, based on a study of survey data from different countries, that there was a consistent relationship between a population’s average level of alcohol consumption and its level of problem drinking. Thus, in order to reduce consumption by the heaviest drinkers, the model suggests that a reduction of the total amount of alcohol use in the country is needed. This idea is integrated in the Norwegian prevention policy, were the overall goal is to reduce alcohol use in the entire population, by means of limiting availability and reducing demands (Meld. St. 30, 2012). Among the main strategies we find high alcohol taxes, restricted access in general where only government-owned companies are allowed to sell wine and spirits (the state Wine Monopoly), age limits for buying alcohol, laws against providing minors with alcohol, and a total ban on alcohol advertisement. This strict alcohol control policy is also reflected in the traditionally low level of alcohol consumption in Norway (Babor, 2010). Norwegian adolescents are at the lower end of the scale regarding alcohol use compared to their European counterparts. According to results from The European School Survey Project on Alcohol and Other Drugs (ESPAD) (Kraus & Nociar, 2016), 19% of Norwegian adolescents reported early onset (at age 13 or younger) and 4% reported early onset of excessive drinking (at age 13 or younger). By contrast, the corresponding rates are 50% and 7% in Denmark, a country in the higher range (Kraus & Nociar, 2016).

Despite the strict and comprehensive alcohol policy, the Norwegian (and Nordic) drinking pattern is characterized by excessive drinking in the weekends. Norwegian adolescents typically drink more on each occasion, but on fewer occasions than for example their counterparts in the Mediterranean countries, who tend to drink more frequently, but drink fewer units of alcohol per drinking occasion (Kraus & Nociar, 2016). It has been argued that
Norway and the other Nordic countries have had a reputation of intoxication drinking since the Viking times, and despite the restrictive alcohol regulation today, the normative climate regarding occasional intoxication in Norway is less restrictive (Nordlund & Østhus, 2013). It is difficult to assess to what extent such “normative climate” influences underage drinking, but it seems evident that norms regarding drinking patterns vary across cultures, and that drinking in the adolescent period vary with cultural characteristics. There is reason to believe that formal and social norms and drinking culture is likely to influence the age of onset, patterns of use and perhaps also predictors and short- and long-term outcomes related to use. Thus, it is necessary to examine predictors and the role of early drinking behaviours for subsequent outcomes in different cultural contexts to get a better understanding of the phenomenon.

1.4 Theoretical perspectives and empirical findings

Over the decades, social scientists have attempted to identify risk and protective factors of early onset and the mechanisms linking early onset to subsequent detrimental outcomes. Central to all theories of child and adolescent development is that the development is based on person-environment interaction and interplay between genetic predispositions and environmental influence. Furthermore, all arenas in life are involved in this developmental possesses and the developmental path is shaped by a complex interplay of factors that promote or inhibit positive development (e.g., Bronfenbrenner, 1979; Sameroff, 2009). This understanding of human development has been formative in the development of theories and empirical models in the study of alcohol use as well. Although theories and empirical studies tend to highlight the importance of applying a multiple risk and protective factors approach in the study of alcohol use, they differ in what influences are considered most important and the degree to which they assume direct or indirect effect between the variables. A main difference has traditionally been between those who seek explanations within a psychosocial framework and those who seek explanations within a genetic and/or biological framework. In recent years, these traditions have gradually converged, and we see more examples of scholars seeking explanations across the traditions. The current thesis is placed within a psychosocial tradition. However, we attempt to take into account that some of the variables included in our models may reflect genetic or biological risk. Our study design is however unfit to determine whether the variables reflect genetic or environmental influence.

In the following, one of the most fully articulated psychosocial framework explaining adolescent involvement in problem behaviour, including adolescent alcohol use, will be
described, namely Jessor and Jessor’s Problem Behaviour Theory (PBT) (Jessor, 1987; Jessor & Jessor, 1977). An account of how the PBT has been formative in the development of empirical models in the field will also be given. Then the focus will turn to theories of potential mechanisms linking early onset of different drinking behaviours to adult outcomes. Findings from empirical studies and recent reviews are presented in relation to these frameworks and some critical knowledge gaps are identified.

1.4.1 Problem behaviour theory
In PBT, problem behaviour is understood as behaviour that is socially disapproved and transgress social and legal norms of the larger society (Jessor, 1987). Early adolescent alcohol use can be understood as such norm-transgressing behaviour. Importantly, adolescent alcohol use is not understood as an isolated behaviour, but rather as a part of a syndrome of problem behaviour. This notion is based on the consistent positive co-variation between adolescent alcohol use and other problem behaviours (Jessor, 1987). However, the theory also stresses that engaging in certain problem behaviours, such as drinking or having sex at a younger age than considered appropriate, may function as a transition-marker affirming maturity and as such be an essential aspect of psychosocial development (Jessor, 1987). So on the one hand, early adolescent alcohol use may be part of a problem behaviour syndrome, and in other circumstances serve a developmental function on the adolescents’ way to adulthood.

According to the PBT, there are five systems that are necessary to explain the development of problem behaviour, including alcohol use, by young people. As originally formulated, the framework comprised only three major systems of explanatory variables; the personality system (e.g., tolerance toward deviance), the perceived environment system (e.g., parental support and controls) and the behavioural system (e.g., general deviant behaviour). The framework has later been modified to include factors from a biological/genetic system (e.g., family history of alcoholism) and social environment system (e.g., peers, school and neighbourhood) (Jessor, 1991). Each system includes variables that reflect either instigations for problem behaviour or controls (protection) against it. The combination of instigators and controls within each of the five explanatory systems generates a profile of psychosocial proneness for problem behaviour. The overall level of proneness across all three systems in the original PBT reflects the degree of psychosocial unconventionality, which implies scepticism towards the values of the broader society, rejection of societal norms, and general readiness for nonconformity (Jessor & Jessor, 1977). The underlying cause explaining involvement in problem behaviours, including alcohol use, during adolescence is thus the
general tendency towards unconventionality, either in the youth’s personality or characteristics in the surrounding social environment (Donovan & Jessor, 1985; Jessor, 1991). Recent reach has shown the utility of the framework in predicting early onset of drinking (Donovan & Molina, 2011).

Most empirical studies of predictors of early onset of drinking and excessive drinking, or studies of their role for subsequent outcomes, do not incorporate the PBT framework directly. However, the majority focus on identifying risk and protective factors within the different systems described in the framework; personality, perceived and social environment, other problem behaviours and biology/genetics. As our strategy to expand the knowledge of predictors of EOI (Paper 1) was to examine whether factors previously identified as key predictors of EOD also predicted EOI, the classification of potential risk and protective factors within different domains of influence provides a useful point of departure. To the extent that it has been possible, in terms of data material, research questions and analytical strategies, we have incorporated variables that reflect the different domains of influence in the second and third studies as well (Papers 2 and 3).

1.4.2 Risk and protective factors

Concepts of «risk» and «protective» factors are often used in this field of research without delineating specific criteria for them to qualify as risk or protective factors. The terms are used in both cross-sectional (e.g., Cleveland, Feinberg, Bontempo, & Greenberg, 2008) and longitudinal designs (for a review, see Donovan, 2004). The basic idea of a risk factor is that it is associated with increased probability of the outcome in question. Statistically this means that there needs to be a statistically significant relationship between the explanatory variable and the outcome variable. Cross-sectional studies are able to establish such associations. However, in addition to establishing an increased probability of the outcome in question, it has been recommended that the temporal nature of the relationship needs to be clarified. More specifically, in addition to establishing an association with an increased probability of the outcome, a risk factor also needs to antedate the outcome in question (e.g., Haggerty & Mrazek, 1994). For this purpose, a longitudinal design is required. In the studies comprising this thesis, this latter view of a risk factor is used. Conversely, a protective factor is the opposite of a risk factor, namely a variable that precedes and is associated with a decreased probability of a negative outcome.
More substantially, although risk and protective factors are identified in statistical terms, there is still a question of whether such factors should be understood as causes or as mere prospective predictors over time. In the present thesis, the terms risk and protective factors are used to indicate prospective predictors over time. Some of the included factors in the models may play a casual role, but this is difficult to verify in a longitudinal design as the one used in this thesis. However, in some cases, there is reason to believe, based on previous theory-driven and evidence-based research, that some of our findings may provide information about the possible causal role of these factors. This will be discussed in chapter 5. The following sections present empirical findings from studies of risk and protective factors of early onset of drinking and excessive drinking, categorized under the different domains of influence described in PBT.

The personality system encompasses variables describing inherent instigators and controls against early onset. One such set of variables are adolescents’ beliefs, attitudes, expectancies and values. Empirical studies of adolescents have identified more tolerant attitudes towards deviant behaviour in general (Brook, Whiteman, Gordon, Nomura, & Brook, 1986) as well as positive alcohol expectancies (Janssen, Treloar Padovano, Merrill, & Jackson, 2018) as prospective predictors of early onset. Higher expectancies for positive effect of consuming alcohol have also been identified as a prospective predictor of early onset of excessive drinking (Jester et al., 2015). Another important set of variables within the personality system is often referred to under the term behavioural under-control. It has been suggested that all sorts of problem behaviours in part can be explained by an underlying deficit in the ability to control impulses (Krueger et al., 2002). As such, adolescents who are less able to control their impulses would be more likely to start drinking alcohol at an early age. Empirical studies have shown that inadequate emotional and behavioural self-control is related to early onset (Wills et al., 2001; Zucker et al., 2008). Temperamental traits are also involved in self-regulation and may also predispose for involvement in risky behaviours, including early alcohol use. For example, greater impulsivity (McGue, Iacono, Legrand, Malone, & Elkins, 2001) and sensation seeking (Webb, Baer, McLaughlin, McKelvey, & Caïd, 1991) have been identified as key predictors of early onset, whereas a shy and inhibited temperament may be a protective factor (Fleming, Kellam, & Brown, 1982; Kerr, Tremblay, Pagani, & Vitaro, 1997). In the studies in this thesis, we have included childhood temperamental traits (Paper 1) and impulsivity/hyperactivity (Paper 2) in the models to cover some of the important elements that are theoretically and empirically linked to early onset of alcohol use in the personality system.
The perceived environment system describes variables reflecting models for conventional or deviant behaviour. Parents and friends are important models during childhood and adolescence. During adolescence, however, the influence from peer group becomes particularly salient (Catalano, Kosterman, Hawkins, Newcomb, & Abbott, 1996). The idea behind models for alcohol use is that parents’ and friends’ attitudes towards alcohol use and concrete behaviours may contribute to shaping the adolescents’ values and expectations towards alcohol use, and as such increase the risk of onset. Empirical studies have identified parental favourable norms towards alcohol as a predictor of early onset (Hawkins, 2002). Affiliation with substance-using peers, or peers with antisocial behaviour, is, furthermore, repeatedly identified as key predictors of early onset (Donovan, 2004; Scholes-Balog et al., 2013; Trucco, Colder, & Wieczorek, 2011). There are, however, uncertainties as to whether there is a direct effect of peer pressure on adolescents alcohol initiation, or whether adolescents tend to seek out friends who are similar to themselves regarding values, beliefs and attitudes towards alcohol (for a review, see Leung, Toumbourou, & Hemphill, 2014). There is probably a bidirectional relationship between peer and adolescent alcohol use (Curran, Stice, & Chassin, 1997; Jessor, 1991). To cover some key elements in the perceived environment system that have also been identified as key predictors of early onset, the studies in this thesis include variables measuring friends’ substance use and antisocial behaviour (Papers 1 and 2).

The social environment system describes the characteristics of the family and the broader social context, such as schools and neighbourhoods. There is extensive empirical evidence that people from socially disadvantaged backgrounds (e.g., low socio-economic status) are at greater risk of alcohol-related problems and mortality (e.g., Poulton et al., 2002; WHO, 2018). There is less evidence regarding the relationship between socio-economy and the timing of alcohol initiation, and the findings are inconclusive (Donovan, 2004; Melotti et al., 2013). Still, we have included indicators of socio-economic status in all our studies (Papers 1-3). Empirical studies have also highlighted the quality of the family environment (e.g., the parent-child relationship) and parenting practices (e.g., strictness versus permissiveness) as important family characteristics within the social environment domain. In general, adolescents with permissive parents tend to engage in higher levels of problem behaviours (Jessor & Jessor, 1977). In relation to early onset, lower parental monitoring and an adverse home environment are found to be important predictors (Donovan & Molina, 2011; Rose, Dick, Viken, Pulkkinen, & Kaprio, 2001). In our studies we have included
variables reflecting different parenting practices (Paper 1) and poor family management (Paper 2) as measures of the adolescents’ social environment.

The *behavioural* system describes involvement in different problem behaviours and also poor school work. There is strong empirical evidence that adolescent alcohol use and other risk-taking behaviours (e.g., smoking and risky sexual behaviour) tend to cluster together (for a review, see Marshall, 2014). Prior involvement in delinquent behaviour has also consistently been identified as a prospective predictor of early onset (for a review, see Donovan, 2004). In our studies, we have included several indicators of problem or risk-taking behaviour, including conduct problems (Papers 1-3), early sexual intercourse (Paper 1), smoking (Paper 1) and grades in school (Paper 3). Some of these behaviours describe externalizing problems, and may be manifestations of a common genetic vulnerability for behavioural under-control, as described in the *personality* system.

Family history of alcoholism is placed under the *biology/genetics* system in PBT, but can also to a certain extent be considered as an environmental influence. Empirical studies have found that children of alcoholics are more likely to abuse alcohol themselves. Several genetically informed studies have furthermore demonstrated that certain individuals have a genetic predisposition to developing alcohol problems (for a review, see Agrawal & Lynskey, 2008). Parental alcohol abuse may thus reflect a genetic vulnerability also inherent in their child, and information about the family history of alcohol problems thus becomes an important control variable in studies on the role of early onset. There is also growing evidence from twin studies that genetic factors may play an important role in explaining adolescents’ initiation of alcohol as well as the susceptibility to alcohol abuse and dependence (Ystrom et al., 2014). A meta-analysis of twin and adoption studies suggest that the heritability of alcohol use disorders is approximately 50% (Verhulst, Neale, & Kendler, 2015). This points to the importance of including family history of alcoholism as a predictor and control variable in studies of predictors of early drinking behaviours and their association to adult outcomes. We have included indicators of alcohol problems in the family in all our studies (Papers 1-3).

### 1.4.3 Proposed mechanisms explaining the association between early onset and adult alcohol problems

Repeatedly, researchers have observed associations between early onset and subsequent high levels of alcohol consumption, alcohol-related problems and alcohol misuse and dependence. Such associations have often been interpreted as causal, i.e., that early onset leads to adult alcohol problems. Suggested mechanisms have been that early onset of drinking may result in
impeded coping strategies, altered neurological functioning, new behavioural repertoires or identities, negatively affected social relationships, or greater tolerance to alcohol (DeWit et al., 2000; Eliasen et al., 2009; Guttmannova et al., 2011; Pedersen & Skrondal, 1998). More recently, several empirical studies have questioned such causal inferences, suggesting that there is more evidence to support the idea that early onset should be understood as a general marker of risk rather than a specific, independent risk factor (King & Chassin, 2007; Prescott & Kendler, 1999; Rossow & Kuntsche, 2013; Ystrom et al., 2014). Below I will give an account of some of the hypothesized mechanisms that have been put forward based on empirical findings of the relationship between early onset and subsequent alcohol-related outcomes.

It has been suggested that late childhood/early adolescence is a particularly sensitive developmental period. Initiation to alcohol in this sensitive period may interfere with biological processes instrumental for healthy functioning, such as for example the development of coping strategies. Such an explanation has been put forward by for example DeWit and colleagues (2000), who in a cross-sectional, community based study of lifetime drinkers (aged 15 and above), found a rapid progression to alcohol-related harm among those reporting having their first drink at ages 11-14. 10 years after the first drink, around 10 to 16% of the subjects met the criteria for a diagnosis of alcohol abuse or dependence. This pattern was not observed among the older starters (19 and older), where only 1 to 2% met the criteria for such diagnosis 10 years after their first drink. Unexpectedly, a delay in progression to harm was observed among the earliest starters (ages 10 and under), suggesting a nonlinear effect of age at first use. Based on these findings, the authors suggest that ages 11-14 is a particularly sensitive period for the onset of drinking that greatly heightens the risk of developing alcohol disorders. The proposed mechanism behind this pattern of findings is that alcohol initiation during this critical developmental period may interfere with important developmental processes (e.g., the formation of self-concept and acquisition of social skills), which are instrumental for healthy functioning. They argue that these changes impede the adolescents’ coping strategies and as a result the adolescents adopt a frequent and heavy drinking pattern as a strategy of coping with stressors and problems (DeWit et al., 2000). The exact nature of this interruption, whether it is of a social or biological nature or a combination, remains obscure. However they emphasise that the sensitivity of this developmental period, because of the many social and psychological changes taking place, is of crucial importance.

This sensitive-period hypothesis, has been tested by Guttmannova and colleagues (2011). In a longitudinal panel study of youths (10 to 33 years) recruited from 18 Seattle
public elementary schools in high-crime areas, they found no evidence that one adolescent period is more sensitive than others. However, all age groups that started drinking regularly before age 21 had a greater rate of alcohol dependence in adulthood. Furthermore, onset before age 11, compared with onset in early adolescence, was related to increased chronicity of adult alcohol dependence. In line with DeWit et al. (2000), the authors suggest that alcohol use in adolescence may compromise socio-emotional, biological and cognitive development which is important for the development of healthy functioning, and thus leads to an increased likelihood and more chronic occurrence of alcohol dependence in adulthood (Guttmannova et al., 2011). There is some support in neurological research that the adolescent brain is particularly sensitive to external influence because of the many alterations in brain function and structure during this period. Particularly the areas modulating sensitivity to a variety of alcohol effects undergoes changes during this period, potentially increasing the predisposition for relatively high levels of adolescent alcohol use, which in turn may set the stage for later alcohol use disorders (for a review, see Patia, 2011).

The interference with adolescent development has also been proposed to be of a purely social nature. For example, in a six-year prospective study of Norwegian adolescents (13-19), Pedersen and Skrondal (1998) found that age of alcohol debut had an independent effect on future alcohol consumption and development of alcohol-related problems. Based on these findings, they suggest that alcohol debut leads to changes in the behavioural repertoire, relations and identity or role, which may explain the development of alcohol-related problems. The suggested mechanism causing these changes is that “(…) something happens to people when they start engaging in certain behaviours. They change, in their own eyes and those of others (…) when the debut takes place before other adolescents take part in the same behaviour” (Pedersen & Skrondal, 1998: 40). This explanation derives from what is described as a social influence loop, where social relationships affect each other negatively, so that the onset of drinking may introduce the adolescent to contexts containing greater exposure to risk factors, for example substance-using peers, which further promote and sustain patterns of use (Jessor, 1991). There is empirical evidence supporting such a theory, as associations with antisocial and substance-using peers have been identified as one of the most robust predictors of early onset (for a review, see Donovan, 2004) and later alcohol-related problems (e.g., Ellickson, Tucker, Klein, & McGuigan, 2001).

A simpler theory that has been put forward is the alcohol habituating theory, suggesting that adolescents initiating alcohol at an early age, progress faster to a more frequent drinking pattern with the consequence of developing a heavier consumption pattern
because of habituation (Eliasen et al., 2009). The adolescents who have an early initiation to alcohol use, have thus had more time to develop a heavy consumption pattern compared to those initiating in late adolescence.

Common for the above-mentioned empirical studies is that they suggest postponement of age of onset as an appropriate measure for prevention of adult alcohol problems, and as such imply elements of causality. The identification of causal mechanisms, however, requires a possibility to establish that a) the cause precede the effect, b) the cause and effect are related and c) there are no other plausible explanations for the effect (Shadish, Cook, & Campbell, 2002). Previous critical reviews of the early-onset literature have concluded that the empirical evidence for causal inference is weak (Maimaris & McCambridge, 2014; Rossow, 2006). The main critique of studies on early onset and its association to adult outcomes, is that early onset is based on retrospective reports and that most of them do not rule out possible confounders (for reviews, see Kuntsche et al., 2016; Rossow, 2006). Although there may always be factors not accounted for, to get a better understanding of the role of early onset for later alcohol problems, prospective studies from early adolescence to adulthood, with adequate control for potential confounders, are needed.

Empirical studies applying such a design are still relatively rare, and the findings are diverging. Some however, give rise to alternative explanatory models than the causal one. For example, in a prospective cohort-study of US children of alcoholics and matched controls, followed from adolescence to young adulthood (ages 11 - 29), King and Chassin (2007) found that early onset (< 13 years) was unrelated to the odds of alcohol and drug dependence when correlated risk factors, such as parental psychopathology, family environment and externalizing symptoms, were taken into account. The authors suggest that these findings support the notion that early initiation is a marker of a genetically determined vulnerability to alcoholism, but not a factor that independently influence such detrimental developmental pathways. In a similar vein, Rossow and Kuntsche (2013) found, in a prospective study of Norwegian adolescents followed from age 13 to 27, that the association between early onset (< 14 years) was confounded by adolescent conduct problems. However, among high-risk individuals (those who had both early onset and conduct problems), there was a significant association between early onset and young adult hazardous drinking. The authors take these pattern of findings as further support for the marker hypothesis, and they suggest that early onset is a component of a problematic lifestyle in general, rather than a specific risk factor for substance use later in life (Rossow & Kuntsche, 2013).
On the other hand, Buchmann et al. (2009), tested the marker hypothesis in a prospective study of a German birth cohort, following adolescents from age 15 to 19. They found that the early onset – young adult hazardous drinking association, cannot solely be attributed to shared genetic and psychopathologic risk. A significant association remained after comprehensive control for possible confounders, including family adversity, parental alcohol use, childhood psychopathology, stressful life and relevant genotypes. The authors take these findings as evidence that early initiation of alcohol use constitutes an independent risk factor for alcohol-related problems in later life, implying causality. The suggested mechanism echoes that of DeWit et al. (2000), namely that an early age at drinking onset promotes the development of inadequate coping strategies, characterized by excessive alcohol drinking when faced with stress.

Within the genetically informed studies, such as analyses of twins, the findings are also diverging. Some show that there is substantial genetic influence on early onset and alcohol use disorders in adulthood, leading to the conclusion that the relationship is of a non-causal nature (Prescott & Kendler, 1999; Ystrom et al., 2014). For example, in a population-based study of Norwegian twins aged 19 to 36, Ystrom and colleagues found that genetic risk for early onset of excessive drinking accounted for 44% of the total risk for alcohol use disorders. Possible mechanisms include heritability of alcoholism and genetic risk of externalizing behaviours (Ystrom et al., 2014). On the other hand, Irons, Iacono & McGue (2015), found in a longitudinal study of twins assessed at target ages 11, 14 and 24 years, that early alcohol exposures predicted adult alcohol problems and related outcomes, despite stringent adjustment for measured and non-measured sources of potential confounding from genetic, shared environmental and non-shared environmental influences. Although the authors conclude that the findings support a causal effect of early alcohol use on adult alcohol problems, they do not necessarily identify the causal mechanism, thereby calling for more research to determine whether these apparent causal effects are authentic.

Thus, it seems that the role of early onset for the development of adult alcohol problems is still not fully understood. There is growing empirical evidence to suggest that early onset is better understood as a general marker of risk rather than a specific, independent risk factor. More specifically, early onset may be a part of a broader risk pathway, where early onset merely reflects a common underlying risk associated with both the risk of early onset and the risk of detrimental outcomes later in life. The diverging results however, calls for more high-quality longitudinal studies examining the role of early onset.
1.4.4 Proposed mechanisms explaining the association between early onset and other life outcomes

Most of the empirical studies on consequences of adolescent alcohol use in general, and early onset in particular, examine adult alcohol-related outcomes. Few have examined early adolescent alcohol use in relation to other important adult life outcomes, such as adult health, psychosocial consequences or educational or work-related outcomes. A recent review summarizing longitudinal studies of adolescent drinking and subsequent outcomes showed that though some studies suggest adverse outcomes with regard to adult health and psychosocial consequences, the existing evidence is of insufficient quality to draw firm conclusions (McCormídge et al., 2011). The evidence for the relationship between early adolescent alcohol use and work-related outcomes are even sparser (for a review, see Marshall, 2014). The current thesis follows up on this reach body and focuses on the association between adolescent alcohol use and educational and work-related outcomes in addition to alcohol related outcomes.

The mechanisms behind the association between adolescent alcohol use and educational and work-related outcomes have not been examined or clarified in any detail. Some suggestions have been made in relation to detrimental work-related outcomes, namely that early-life alcohol use may influence a certain “life career”, e.g., involvement in crime or substance use, which may prevent them from getting or maintaining a job (Sidorchuk, Hemmingsson, Romelsjö, & Allebeck, 2012). More evidence is found on the relationship between chronic adolescent alcohol and drug use and lower educational attainment, where it has been suggested that substance use may increases absenteeism, the risk of dropping out of school, and result in poor school performance, which in turn may diminish their subsequent chances of finding work (for a review, see Henkel, 2011). Although there are few systematic studies on how alcohol use in adolescence is prospectively related to educational and work related outcomes later in life, it seems that particularly early and heavy alcohol use is related to negative educational and work related outcomes (Ellickson, Tucker, & Klein, 2003; Henkel, 2011; Keng & Huffman, 2007; Renna, 2007). Research on alcohol use in the adult years and its association with work related outcomes draws a slight different picture. Generally, moderate consumption has been associated with higher income (Barrett, 2002), lower risk of unemployment, sickness-absence and disability pension awards (Jørgensen, Thygesen, Becker, & Tolstrup, 2017). Suggested mechanisms have been that moderate drinking improves the individual’s health and hence productivity and earnings (Barrett, 2002),
or alternatively, that moderate drinkers, compared to abstainers and those reporting alcohol-related problems, are in better health in the first place (Ormond & Murphy, 2017).

The general lack of systematic studies of the relationship between early drinking behaviours and adult life outcomes other than alcohol use warrant further investigation of the role of early adolescent alcohol use for important life outcomes, such as educational and work-related outcomes. Furthermore, the mixed pattern of findings in relation to the potential positive effect of alcohol use in adulthood and negative effect of alcohol use in adolescence, warrant further investigation of the age-specific aspects of alcohol use. These topics are addressed in the third study (Paper 3) in this thesis.

1.5 Summary of knowledge gaps
The review of the literature has revealed critical knowledge gaps in several areas. First, major research efforts over several decades have resulted in substantial knowledge on risk and protective factors for early onset of drinking. However, although a growing number of studies suggest that greater attention should be paid to early onset of excessive drinking, as it seems to play a more important role in the course of negative development, few have examined possible risk and protective factors of early onset of excessive drinking. This is part of the focus of the present thesis. To gain more knowledge on risk and protective factors of early onset of excessive drinking, our point of departure was to identify key predictors of early onset of drinking as identified in the literature, and to examine whether these also predict excessive drinking. Furthermore, in order to identify robust predictors of excessive drinking, it is important to include variables from all of the different domains of influence described above. The first study in this thesis (Paper 1) is using a long-term longitudinal study with a variety of variables from these different domains to examine predictors of early onset of both drinking and excessive drinking.

Second, despite the many empirical studies that have examined the relationship between early onset and adult alcohol-related outcomes, there are still some contrasting results with regard to the role of early onset that call for further examination. In particular, important limitations of previous research needs to be addressed, by examining whether early adolescent drinking behaviours prospectively predict risk of alcohol-related problems even after adjustment for a wide range of potential confounders. Furthermore, few have examined whether early onset of more than a few sips of alcohol is predictive of later alcohol problems or whether only the consumption of more excessive amounts of alcohol in early age is related to such risk. Moreover, as the cultural context is likely to influence which factors are related
to onset as well as patterns of use, it is necessary to examine adolescent drinking behaviours in different cultural contexts. To our knowledge, few have applied a cross-national design predicting hazardous drinking in late adolescence/young adulthood from early and excessive adolescent drinking. This is the focus in the second study (Paper 2).

Third, the majority of empirical studies on adolescent alcohol use focus on the association with alcohol-related outcomes in adulthood. There is less knowledge about educational and work-related outcomes. We follow up on the call for more high-quality longitudinal studies focusing on the association between early alcohol intoxication and education, income and labour market integration in adulthood. Moreover, the mixed pattern of findings, where alcohol use in adolescence demonstrate potential negative effects and adult alcohol use demonstrate potential positive effects, further calls for a systematic investigation of how the prospective association of alcohol use with education and work outcomes vary according to age. This is part of the focus in the third study (Paper 3).
2. Aims and research questions

The overall aim of the thesis is to contribute to a better understanding of predictors of early onset of drinking (EOD) and excessive drinking (EOI) and the prospective relationship between EOD/EOI/ alcohol intoxication (AI) and important life outcomes. Such knowledge can inform health promotion, prevention and early intervention efforts. Data from three population-based longitudinal studies, together with registry data, were used to examine the following main research questions:

1) Which factors prospectively predict EOI and do these predictors differ from those predicting EOD? (Paper 1)

2) Is EOD, independent of EOE, prospectively related to hazardous drinking in late adolescence/young adulthood in Norway and Australia, two countries with different prevention policies and drinking cultures? (Paper 2)

3) How is AI at different ages from early adolescence to young adulthood associated with subsequent outcomes with regard to education, income and possible risk of unemployment and disability? (Paper 3)
3. Methods

3.1 Samples and procedures

The papers in this thesis are based on data from three population-based longitudinal survey studies. The first and second papers (Papers 1 and 2) are based on data from the Tracking Opportunities and Problems study (TOPP). The second paper (Paper 2) is additionally based on data drawn from the Australian part of the International Youth Development Study (IYDS). The third paper (Paper 3) uses data from the Yong in Norway Longitudinal study (YiN) linked to register data. A description of setting, sample and data collection procedures is outlined below.

3.1.1 The TOPP study

The TOPP study is a population-based prospective study of Norwegian children and their families, followed over an 18-year period from 1993 to 2011. The initial study was set up to provide knowledge on precursors and developmental pathways to good mental health and adjustment problems among young children and their parents. Over the years the study has expanded and now include a wide range of topics relevant for adolescent health and development as well as important aspects related to the parents, such as parenting, parents’ physical and mental health, sick leave and partnership.

The recruitment was set out to resemble the overall population in the south-eastern part of Norway at the time. In Norway, more than 95% of families attend scheduled routine check-ups at child health clinics during the first five years of the child’s life, and was therefore chosen as the setting for recruitment. All children and their families (n = 1,081) from 19 public child health clinics across 6 municipalities (28% living in large cities, 55% in densely populated areas and 17% in rural areas) were invited to the study. The first data were collected when the families attended their toddlers’ 18-month check-up. The only inclusion criterion in the study was Norwegian language ability (2% of all families were excluded based on this criterion) (Mathiesen, Sansnon, & Karevold, 2018; Nilsen et al., 2017).

The participants have been followed up over eight data collections from when the children were 1.5 years (t1 in 1993) to 18.5 years (t8 in 2011). Questionnaires were handed out to the mothers and returned at the clinic in the three first waves. The remaining surveys were conducted by mail. The respondents were also offered the option to respond to a web-based version of the survey (t7-t8) or by telephone interview by trained assistants (t8 only). In the first four waves, when the children were 1.5, 2.5, 4.5 and 8 years respectively, the questionnaires were only given to the mothers. From age 12.5 (t5) and thereafter, the
adolescents as well as their mothers replied to their separate questionnaires. In this thesis, data from t1-t6 were used in Paper 1 and from t6 and t8 in Paper 2 (see Figure 1).

At t1, 85% (n = 913) of the invited mothers participated. Background data from the child health clinics at 1.5 years showed that non-respondent mothers did not differ significantly from responding mothers in age, education, employment status or marital status (Mathiesen & Tambs, 1999). The overall response rates at the different waves of data collection, as well as the subsamples used in the studies comprising the current thesis, are depicted in Figure 1. In the first study (Paper 1) we drew a subsample consisting of all adolescents who had answered the questions about drinking and intoxication experiences at t6 (age 14.5), and where they themselves and their mothers had answered the questionnaires at t5 (age 12.5), where most of the predictors were derived. There were four respondents who reported that they had been intoxicated at t5. As our aim was to predict EOD without EOI at t6, these 4 respondents were not included in the final subsample. In total, adolescent and mother reported data from 382 participants comprised the sample used in the first study (Paper 1). In the second study (Paper 2) a subsample was obtained from surveys of the participants and their mothers conducted in 2006 (t6, age 14.5) and 2011 (t8, age 18.5). In the final sample, data at both time points of interest were available for 329 young people.

Previous attrition analyses have shown that low maternal educational level at baseline predicted maternal dropout from t1 to t5 (Karevold, Roysamb, Ystrom, & Mathiesen, 2009) and to t7 (Gustavson, von Soest, Karevold, & Roysamb, 2012). No differences in correlations between 15 baseline variables (t1) were found among those who stayed until t7 and those who dropped out (Gustavson et al., 2012). Similarly, adolescent attrition from t1 to t7 and t8 was predicted by low maternal education level, but also by male gender (Kjeldsen et al., 2016; Nilsen, Gustavson, Roysamb, Kjeldsen, & Karevold, 2013).
Figure 1. Number of participants in the different waves of data collection and subsample in Papers 1 and 2

Notes: t1 to t5 is mother-report about their children (m) and t5 to t8 is adolescent self-report (a). The response rate at t1 is calculated on the basis of the number of invited families (n=1,081). All the following response rates are calculated on the basis of the number of mother-reports about their children at t1 (n=939).

3.1.2 The IYDS
The IYDS is an ongoing binational longitudinal study initiated in 2002 of three cohorts (t1 included children in grade 5, grade 7 and grade 9) of young people in Victoria, Australia and Washington State, United States. The IYDS examines the development of adolescent behaviours, including antisocial behaviour and substance use, and assesses a broad array of risk and protective factors within the adolescents’ individual, peer, family, school, and community domain (McMorris, Hemphill, Toumbourou, Catalano, & Patton, 2007). The study was set up to examine the development of healthy and problem behaviours among young people. In this thesis, only data from the Australian arm of the study are used and described, as data on young adults in the US arm of the study were not available at the time of analysis.

The IYDS is a school-based survey with a state-wide representative sample recruited and surveyed in 2002 and followed up in 8 subsequent waves. Participants were recruited using a 2-stage cluster sampling approach for schools and students. In the first stage, schools were randomly selected, and in the second stage, a target classroom within each school was randomly selected. Public and private schools containing grades 5, 7, or 9, were randomly selected using a probability proportionate to grade-level size sampling procedure (Kish, 1965). At t1, the surveys were administered by study staff and were group-administered in
classrooms during a 50- to 60-minute period. Students absent from school were administered surveys later under the supervision of trained school personnel or in a small percentage of cases (less than 3%), over the telephone by study staff (McMorris et al., 2007). Students were followed up annually for three consecutive years with 98% retention (Mason et al., 2011), and thereafter the cohorts were surveyed at different intervals, still with over 80% retention on all waves from 2002 to 2014. Extensive locating and tracking procedures were in place to ensure these retention rates (e.g., contacting schools, calling the family’s home/work/mobile, checking the electoral roll, contacting people of the same surname in a similar geographic location using phone directories) (Scholes-Balog et al., 2013).

The second study (Paper 2) used data from the IYDS in addition to data from the TOPP sample. The oldest age cohort (grade 9) in the IYDS was used, as this best matched the ages at which the TOPP data collection was conducted. In the oldest cohort, 1,288 students were eligible to participate, of whom 973 (76%) participated at t1 and 788 in the follow-up at t7 (81% retention). Honesty criteria based on responses to the t1 survey was used to remove 2 participants and so the final sample with data at both time points was 786. The data from the surveys included in this study were conducted in 2002 (t1, mean age 15) and 2010 (t7, mean age 23). The IYDS also includes parent report on socio-economic status from a phone survey conducted in 2002.

3.1.3 The YiN study
The YiN is a population-based survey study of Norwegian students in grades 7–12 with data collected at four time points: 1992 (t1), 1994 (t2), 1999 (t3), and 2005 (t4). The study was the first nationwide study of adolescents in Norway. The study covers several aspects of development, with a main emphasis on psychological, psychiatric, sociological and pedagogical variables (Wichstrøm, 2002). The initial sample comprised 12,287 students in grades 7–12 (aged 12-20) from 67 junior and senior high schools in Norway, of whom 97% agreed to participate.

The sampling procedure was designed to obtain a representative sample of the student population in Norway. At the time of the start-up of the study, 98.5% of all Norwegian 13- to 16-year-old adolescents attend junior high schools, and 97% continue to senior high or vocational school. As such, the sample was drawn from a population comprising almost the entire age cohort. First, all schools in Norway were divided into 5 geographical strata. Second, the junior high schools (grades 7-9) were stratified by school size (3 strata) which is closely related to the urban-rural dimension in Norway. At the high school level, the school...
size does not correspond to the degree of urbanizations. However, there is a clear distinction between academic and vocational high schools, in addition to schools with both lines of study. High schools were therefore stratified in accordance to these three lines of study (3 strata). Participating schools were then drawn according to school size and strata. Each school’s sampling probability was proportional to the number of students enrolled in the school, thereby ensuring that the probability of being selected to participate in the study was equal for all students in Norway (Wichstrøm, 2002). In order to avoid the possibility of students influencing each other’s responses, all students in the participating schools completed the questionnaire simultaneously during two consecutive school hours. At t1 and t2 the questionnaires were distributed and completed in the classroom, while at t3 and t4 the participants received the questionnaires by mail. At t4 the respondents could also choose an online questionnaire or telephone interview. At t4, the respondents were furthermore asked for their consent to link the data to several registers.

Three schools were included at t1 for non-prospective reasons and were not part of the follow-up. In two other schools, the project identification records were lost due to a burglary in the schools’ archives. At t2, students who still attended the same school as at t1 were followed up with questionnaires in school. Since a sizable portion of the students had completed their three-year track in the junior or senior high school that they attended at t1, participants no longer in their original school at t2 received the questionnaire by mail. The response rate of those still attending the same school at t2 was 92%. Only students who completed the questionnaire at school at t2 (n = 3,844) were followed up at t3 because of the comparatively lower response rate among those receiving the questionnaire by mail. Because the study was originally planned to be a two-wave study, new informed consent had to be obtained at t2. Those then consenting (n = 3,507; 91%) received questionnaires by mail at t3 and t4, with data received from 2,924 (84%) and 2,890 (82%) participants, respectively. At t4, the respondents were asked for their consent to link the data to several registers, to which 2,602 respondents (90%) agreed. The overall participation rate of the final sample, based on all eligible students at t1 who still were at their original school at t2, was therefore 68% at t3, 67% at t4, and 60% concerning assessment of register data.

Previous attrition analyses have showed that drop-out at later waves were predicted by being male, poor grades, being in vocational training, conduct problems, low parental socio-economic status, few hours spent on homework and degree of urbanization (Wichstrøm, 2000a, 2000b; Wichstrøm & Hegna, 2003).
In the third study (Paper 3), only data from students born between 1974 and 1979, and who had agreed to register linkage (von Soest, Bramness, Pedersen, & Wichstrøm, 2012; von Soest, Wichstrøm, & Kvalem, 2016) were included (n = 2,602). These students were 13 to 18 years of age at t1 and 27 to 31 years of age at t4.

3.2 Ethics

Data collection and permission to undertake data analysis was approved by the Norwegian Data Protection Authority and the Regional Committees for Medical and Health Research Ethics (REC) South East Norway in the TOPP and YiN study. Participants in YiN provided written consent. The TOPP study started in 1992/1993 when written consent in research studies was not a requirement. However, in later years, REC has reviewed all information given to participants and concluded that the study fulfills essential requirements for informed consent. Study participation was voluntary. Information about the study, the possibility to skip questions, and the right to withdraw from the study at any point, was provided in writing to both the children and their mothers for each data collection.

In the Australian IYDS study, ethics approval was first gained through the Ethics in Human Research Office at the Royal Children’s Hospital in Victoria. At time 1, permission was then obtained from the Victorian Department of Education and Training for government schools and the Catholic Education Office for some private schools and then by school principals. Parents provided written consent for their adolescent to participate in the study and adolescents provided assent to complete the survey. At time 7, ethics approval was obtained through The University of Melbourne Human Ethics in Research Committee and young adult participants provided online assent at the time of survey completion. The study was approved by the Royal Children's Hospital Ethics in Human Research Committee.

3.3 Measures

Data from different ages were used in all analyses, and both mother-report and adolescent self-report were used, in addition to register data. We used data from the Norwegian TOPP study in the first and second studies (Papers 1 and 2). In Paper 1, analyses included independent variables measured from t1-t5 (child age from 18 months to 12.5 years) and dependent variables from t6 (age 14.5). In paper 2 we used independent variables measured at t6 (age 14.5) and dependent variables measured at t8 (age 18.5). Data from the Australian IYDS were included in Paper 2, where independent variables were measured at t1 in the oldest cohort (age 15) and dependent variables measured at t7 (age 23). In the third paper,
data from the Norwegian YiN study were used. Independent variables were measured from t1 (ages 13-18) to t4 (ages 27-31) and the dependent variable was collected from public registers when all respondents had reached the age of 32. As most papers include variables within the same domains, they are described by domain rather than by paper.

3.3.1 Alcohol use in adolescence and young adulthood
In the first paper we only used data from the Norwegian TOPP sample and abstinence, alcohol use and intoxication were measured by adolescent self-report on the following two items at 12.5 (t5) and 14.5 (t6) years: “Have you ever tasted more than a few sips of alcohol?” and “During the past 12 months, have you had so much to drink that you felt clearly intoxicated?”. Both questions had five response categories (Never, Once, 2-5 times, 6-10 times and More than 10 times). These items were categorized into one variable with three groups at 14.5 years; 0 - Abstinent (never tasted, never been drunk), 1 - early onset of drinking (EOD; tasted one or more times, but never been drunk) and 2 - early onset of intoxication (EOI; tasted one or more times and been drunk one or more times). This variable constituted the dependent variable in Paper 1.

In the second paper we used questions from both the Norwegian TOPP sample and the Australian IYDS sample. From the Norwegian sample we used the same two items as described above filled in at t6 (age 14.5). Responses were now reclassified into two dummy variables; 1) early onset of drinking (EOD) relative to abstinent (have tasted one or more times, but not been intoxicated in the past 12 months), and 2) early onset of excessive drinking (EOE; tasted one or more times and been intoxicated one or more times past 12 months). The wording was changed from EOI to EOE in this study to better match the AUS measure. In the Australian sample these concepts were measured by adolescent self-report using three items; “In your lifetime, have you ever had more than just a few sips of an alcoholic beverage (like beer, wine or spirits)?” with five response categories (Never, 1 or 2 times, 3 to 5 times, 6 to 9 times, 10 or more times), “Think back over the past 2 weeks. How many times have you had five or more alcoholic drinks in a row?” (None, Once, Twice, 3-5 times, 6-9 times, 10 or more times) and “How often over the past year has your alcohol use caused you to get so drunk you were sick or passed out?” with items rated on an 8-point scale from never to 40 times or more (age 15). Responses were reclassified into two dummy variables; 1) EOD relative to abstinent (one or more times of lifetime alcohol use, but never binged and never sick or passed out), and 2) EOE (one or more lifetime alcohol use and one
or more times binged and/or one or more times sick or passed out). In the second paper these variables across samples constituted predictor variables.

In the third paper we used data from the YiN study and a measure of Alcohol intoxication (AI) from early adolescence to young adulthood as a predictor variable. AI was measured by the question: “During the past 12 months, have you had so much to drink that you felt clearly intoxicated?” with response options 1 – never, 2 – once, 3 – 2 to 5 times, 4 – 6 to 10 times, 5 – 10 to 50 times, and 6 – more than 50 times.

The outcome variable in Paper 2 measured hazardous drinking in late adolescence/young adulthood. In both the Norwegian (age 18.5) and Australian (age 23) surveys, these concepts were assessed using the Alcohol Use Disorders Identification Test (AUDIT) (Saunders, Aasland, Babor, De la Fuente, & Grant, 1993). The AUDIT is a widely used screening tool consisting of 10 items constructed to identify persons with hazardous (i.e., those who are at risk of alcohol-related problems) and harmful (i.e., those experiencing some alcohol-related problems) patterns of alcohol consumption. The scores of the full AUDIT range from 0 to 40, and the generally accepted cut-off for hazardous drinking is 8 and above, and 16 and above for harmful use (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001; Reinert & Allen, 2007; Saunders et al., 1993). In Paper 2, we examined only hazardous use. In the Norwegian questionnaire, item 3 differs from the original AUDIT in that it asks “How often do you have five or more drinks on one occasion” instead of “six drinks” which is the original wording. Cronbach’s alpha for the whole scale was .77 in the Norwegian sample and .83 in the Australian.

3.3.2 Educational attainment and labour market outcomes
In the third paper we used register data from Statistics Norway to obtain information about the following, the year the respondents turned 32: education, income, and whether they had received social or unemployment benefits, labelled unemployed, and disability or rehabilitation benefits, labelled disabled or on rehabilitation. Highest level of education was coded into five categories ranging from 1 (junior high school) to 5 (higher university degree). Gross annual income was recoded into 10 equally sized groups, with values from 0 to 1. Dummy variables show whether participants had ever received social or unemployment benefits and disability or rehabilitation benefits by the year they turned 32.
3.3.3 Individual level variables

In Paper 1 *adolescent temperament* was assessed at 12.5 by maternal report on the EAS Temperament Survey for Children (Parental ratings) (Buss & Plomin, 1984). The scale assesses the adolescents’ degree of emotionality (the tendency to become aroused easily and intensely, 12 items), activity (preferred levels of activity and speed of action, 4 items), sociability (the tendency to prefer the presence of others to being alone, 4 items) and shyness (the tendency to be inhibited and wary in new social situations, 4 items). Responses were rated on a 5-point scale ranging from 1 (*Not typical*) to 5 (*Very typical*). Mean scores were computed and Cronbach’s alpha at 12.5 for the four temperamental subscales were .81 (Emotionality), .82 (Activity), .63 (Sociability) and .77 (Shyness).

In Paper 2 *hyperactivity* was measured at age 14.5 in the Norwegian TOPP sample with a subscale of the Strengths and Difficulties Questionnaire (SDQ) (Goodman, Meltzer, & Bailey, 1998) with five items (e.g., ”I am restless”, “I find it hard to sit down for long”, “I think before I do things”) with four response categories ranging from 3 (Fits very well) to 0 (Doesn’t fit at all) (Cronbach’s alpha = .71). The items closest to this dimension in the Australian IYDS was *impulsivity*, which was measured at age 15 by three items describing typical ways to act (e.g., thinking before acting, rushing into things, answering before thinking). Response options were NO! (1), no (2), yes (3), YES! (4) (Cronbach’s alpha = 0.57).

*Conduct problems* the past year were assessed at age 12.5 by 22 self-reported items (Paper 1) and at age 14.5 by 19 self-reported items (Paper 2), with response options ranging from 1 *Never happened* to 5 *Happened more than 10 times* in the Norwegian TOPP study. In Paper 1 we constructed a mean score (Cronbach’s alpha = .74), and in Paper 2 we transformed the scale to a yes/no variable, creating a sum score ranging from 0 to 18 (Cronbach’s alpha = .84). The scales are based on three different Scandinavian scales of antisocial behaviour and the items cover behaviours like stealing, verbally and physically aggressive behaviours, loitering, vandalism and questions about carrying weapons (Kjeldsen, Janson, Stoolmiller, Torgersen, & Mathiesen, 2014). In the Australian sample, also used in Paper 2, *conduct problems* were measured at age 15 by nine items covering behaviours such as stealing, physically aggressive behaviour, suspension from school, arrests and questions about carrying weapons, selling drugs and being drunk or high at school. The response options ranged from *never* (1) to *40+ times* (8) and a sum index was created (Cronbach’s alpha = 0.64). In Paper 3 we used a 15-item measure of *conduct problems* at t1 (ages 13-18), which approximates diagnostic criteria for conduct disorder in the DSM-III-R (Wichstrom, Skogen, & Osia, 1996).
The conduct problem index consists of the number of problems reported (Cronbach’s alpha = .75). Smoking (Papers 1 and 2) and early sexual intercourse (Paper 2) were included as indicators of early risk behaviours in Papers 1 and 2. Adolescent smoking was assessed by one item with responses ranging from 1 \((\text{Have never smoked})\) to 4 \((\text{Smoke daily})\) in Paper 1 (age 12.5) and in Paper 2, smoking was assessed by a single item asking if adolescents had smoked at least once in their life \((\text{yes/no})\) in both the Norwegian (age 14.5) and Australian (age 15) sample. In Paper 2, early sexual intercourse was assessed by one item asking if the adolescents had ever had sexual intercourse \((\text{yes/no})\) in both the Norwegian (age 14.5) and Australian (age 15) sample. In Paper 3, school grades in Norwegian, Mathematics and English were assessed at ages 13-18 by self-report and mean scores were computed, ranging from 1 \((\text{lowest grade})\) to 6 \((\text{highest grade})\).

Kandel and Davies’ (1982) Depressive Mood Inventory was used as a measure of adolescent depressive symptoms at ages 13-18 in Paper 3, with internal consistency of \(\alpha = 0.78\). This measure was derived from the Hopkins Symptom Checklist (SCL, Derogatis, Lipman, Rickels, Uhlenhuth, & Covi, 1974) and asks for ratings of depressive symptoms during the preceding year on a 3-point scale. In the present version, the response format was changed back to the original format in the SCL, restricting the ratings to the preceding 14 days and applying a 4-point scale with the response options from 1 \((\text{not at all})\) to 4 \((\text{extremely})\). Mean scores were computed, ranging from 1 to 4.

3.3.4 Antisocial peer environment
In Paper 1 Deviant behaviour of friends was assessed at age 12.5 by four items asking the adolescents about the number of close friends who: smoke regularly, use alcohol approximately once a week, have tried cannabis, or have been in contact with the police for illegal activities. The response categories were 1 \((\text{none})\), 2 \((\text{one friend})\) and 3 \((\text{several friends})\). A mean score was computed and used in the analyses. In Paper 2 the assessment of friends’ substance use in the Norwegian sample (age 14.5) was computed by using adolescent-report on how many of their most important friends: “drink alcohol approximately once a week” and; “have tried cannabis, marijuana or other illegal drugs”. In the Australian sample (age 15), friends’ substance use during the past 12 months was indicated by questions measuring how many of your best friends have: “tried alcohol (like beer, wine, or liquor/spirits) when their parents didn’t know”, “used marijuana (pot, weed, grass)”, “used other illegal drugs (like cocaine, heroin, LSD/acid, or amphetamine/speed)”. Dummy variables were constructed in
both samples, contrasting those who reported having at least one friend that has substance use experiences with all the others.

3.3.5 Socio-economic factors

*Family status* was in Paper 1 reported by the adolescents (age 12.5) and recoded into a dummy variable where those living with both biological parents (value 1) were contrasted with adolescents living in all other living arrangements (e.g., living with only mom, only dad, mom/dad and new partner, foster parents) (value 2). Parents’ level of education was used as an indicator of socio-economic status (SES) in all three papers. In Papers 1 and 2, *maternal education* was assessed when the adolescents were 12.5 and 14.5 years of age respectively, by asking the mothers to report their highest level of education on a scale from 1 (*9 years primary school or less*) to 5 (*> 4 years at university or university college*) in the Norwegian sample and by mother-report on a 3-point scale of *Less than secondary school* (1), *Completed secondary school* (2) to *Completed post-secondary education* (3) in the Australian sample when the adolescents were 15 years of age. In Paper 3, information about *parental educational level* was collected using register data when the respondents were 16 years of age and was measured on a 4-point scale ranging from compulsory elementary school only to high university level. The parent with the highest level of education was used. In Paper 1 (adolescents’ aged 12.5), we also included information about mothers’ employment status and household economy. *Employment* measures the mothers’ workforce participation in terms of percentage of paid work (1 = *No paid work*, 2 = < 50%, 3 = 50–80% and 4 = 80–100%). *Household economy* was measured by asking the mothers “How do you/your family cope with your current financial situation?” with five response categories ranging from 1 *We cope very poorly* to 5 *We cope very well*.

3.3.6 Family level variables

Measures of alcohol and drug use in the family were included in all three papers. In Paper 1, *alcohol problems in the household* was defined to have occurred if the mothers reported having experienced “alcohol problems in the household” one or more times during the past 12 months with children aged between 1.5 and 12.5 (0 = No, 1 = Yes). In Paper 2, *alcohol and drug use in the family* in the Norwegian sample was defined to have occurred if the adolescents reported having experienced that “one of the people closest to me uses too much alcohol, pills or other drugs” one or more times during the past 12 months between the ages 12.5 and 14.5 (0=No, 1=Yes). In the Australian sample alcohol and drug use in the family was measured at age 15 by adolescent-report on the question: «Has anyone in your family ever
had a severe alcohol or drug problem?” (0=No, 1=Yes). In Paper 3, heavy parental drinking was assessed at each of the first three data collection waves with the question “Have you ever seen your parents drunk?” The response options ranged from never to a few times a week. The items from all three time points (ages 13-25) were summed to create a parental binge drinking index (values 0–12).

Parenting practices in Paper 1 were measured by mother-report when the adolescents were 12.5 years of age on the Alabama Parenting Questionnaire (Shelton, Frick, & Wootton, 1996), which measures positive involvement with children (Cronbach’s alpha = 0.76), use of positive discipline techniques (Cronbach’s alpha = .70), consistency/inconsistency in the use of such discipline (Cronbach’s alpha = .67) and other disciplinary practices (Cronbach’s alpha = .65). Items were rated on a 5-point Likert scale from 1 (Never) to 5 (Always) and mean scores were computed. We also included a 10-item revised subscale on parental strictness and supervision (mother-report) from the Lamborn Parenting Scales (Lamborn, Mounts, Steinberg, & Dornbusch, 1991), with response categories recoded into “Low” (coded 1) to “High” (coded 3) levels of strictness/supervision. Mean scores were computed and Cronbach’s alpha was .75. In Paper 2, poor family management was in the Norwegian sample assessed using adolescent-report (age 14.5) on the short version of the “Keeping tabs” questionnaire developed for the NICHD Study of Early Child Care and Youth Development (SECCYD) based on the work of Stattin and Kerr (2000). The six items are questions regarding their parents’ supervision and monitoring (e.g., “how much does a parent know about...”; “who you spend time with?”; “...how you spend your money?”; “...where you go after school?”). Responses ranged from 1 to 4 (“knows everything” to “doesn’t know at all”) and indicate the extent to which the parent is thought to know about different aspects of the child’s whereabouts and day-to-day activities (Cronbach’s alpha = .85). In the Australian sample, poor family management was measured by a 9-item scale asking adolescents’ (age 15) agreement to statements such as “My parents ask if I’ve gotten my homework done”, “The rules in my family are clear”, “If you skipped school without your parents’ permission, would you be caught by your parents?”. Response options were: NO! (4), no (3), yes (2), YES! (1) (AUS, Cronbach’s alpha = .77).

3.3.7 Demographic variables

Gender and age were measured.
3.4 Statistical analysis

3.4.1 Multinomial logistic regression
In the first study (Paper 1), predictors of EOI and EOD at age 14.5 were examined by means of multinomial logistic regression analyses. The multinomial logistic regression analyses allow the dependent variable to have more than two categories, in our case we had three: Abstinent, EOI and EOD. Being abstinent was chosen as the comparison group. Thus, each analysis produced two comparisons: the odds of EOI and EOD compared to abstinent. The independent variables in the models were categorized under four domains: temperament, socio-economic factors, family factors and adolescents’ and friends’ behaviour. The analyses were conducted in two main steps. First, all variables were included as predictors one by one in a series of multinomial logistic regression analyses, with control for age, gender and alcohol use before age 12.5. In order to identify predictors that differentiate between EOI and EOD, we conducted additional analyses using EOD as comparison group. Second, we entered all significant predictors from these analyses simultaneously into a multiple multinomial regression analysis. All models were adjusted for age, gender and alcohol use before age 12.5. Participants with intoxication experiences before the age of 12.5 were excluded from the analysis in order to predict EOD and EOI by age 14.5 without including respondents who had been drunk before this age.

3.4.2 Modified Poisson regression analysis
The relationships of adolescent EOD and EOE with hazardous drinking in late adolescence/young adulthood were examined by means of a modified Poisson regression approach where Poisson regression with robust error variances were estimated (Paper 2). This approach has been recommended instead of binary logistic regression analysis because prevalence ratios (PR) or risk ratios are obtained, thereby providing a more intuitive estimate of the association between predictors and outcomes than odds ratios from logistic regression models (Espelt, Mari-Dell’Olmo, Penelo, & Bosque-Prous, 2016; Zou, 2004). Moreover, Poisson regressions can easily be conducted without difficulties converging. In the second study (Paper 2), the two dummy variables for EOD and EOE were simultaneously included as predictors of hazardous drinking (AUDIT =>8) together with age and gender. Moreover, a series of Poisson regression analyses was performed to identify associations between each potential confounder with hazardous drinking, controlling for age and gender. Finally, EOD, EOE and all potential confounders were included simultaneously in Poisson regression analyses to examine the unique associations of adolescent EOD and EOE with late
adolescent/young adult hazardous drinking. To examine the robustness of the results, all the analyses were re-ran using binary logistic models. All continuous predictor variables were standardized. Results thus indicate the change in the outcome associated with one standard deviation change in the predictor.

3.4.3 Latent growth curve modelling
To model developmental trajectories of adolescent AI, latent growth curves were constructed. As respondents were born between 1974 and 1979, respondents were divided into six age cohorts according to their birth year. Multiple group analysis in the framework of structural equation modelling was then used, where each of the six groups was defined by one age cohort. A latent growth model was then estimated in each group. The latent growth curves were based on dichotomous indicators of AI. For this purpose, probit regressions in the framework of latent response variable transformation was used (Masyn, Petras, & Liu, 2014) to transform dichotomous responses into normally distributed continuous variables before estimating the growth curve model (Lee, Wickrama, & O’Neal, 2017). The same latent growth model was constructed in each of the six age cohort groups. Factor loadings for growth factors were parameterized according to individuals’ age, such that common growth trajectories for AI across all six groups were specified (for a more detailed account of the analytical framework, see Duncan, Duncan, & Strycker, 2006; Preacher, Wichman, MacCallum, & Briggs, 2008). Thus, based on multiple group analyses, growth curves were constructed representing development from age 13 to age 31 of the risk of AI. Linear and non-linear trajectories were tested by including linear and quadratic slope factors in growth curve models. We thus estimated three parameters in the most complex growth models: the intercept, estimating initial risk alcohol use at age 13, the linear slope, representing linear change, and the quadratic slope, representing quadratic change in the risk of AI from age 13 to age 31.

We dichotomized AI in four different ways to model risk trajectories for having had at least (a) one, (b) two, (c) six, or (d) eleven episodes of AI during the last twelve months. Four different growth curve models were then constructed based on these four dichotomizations of AI.

Gender differences in trajectories were examined by means of conditional growth curve models by regressing growth factors (i.e., intercept, slope and quadratic growth factor) on gender. Long-term outcomes of alcohol trajectories were examined by regressing indicators of educational and labour marked inclusion on the intercept of AI. For dichotomous
dependent variables, probit regressions were modelled. For continuous outcomes, linear regression was used. As a main aim of the paper was to examine how AI at different ages was related to educational and labour marked outcomes, we estimated different models where the intercept of the growth curves were parameterized in three different ways: (a) the intercept was parameterized at age 13, such that the intercept was indicating the risk for AI at age 13, (b) the intercept was parameterized at age 22, indicating risk for AI at age 22, (c) and the intercept was parameterized at age 31, indicating risk for AI at age 31.

3.4.4 Handling of missing data
In Paper 1, the analyses were limited to those participants who had responded to the questionnaires at t5 (predictors) and t6 (outcome). Three respondents were missing on the outcome variables at t6 and were removed from the analyses. Missing in the predictor variables were handled in different steps. As a first step, we investigated the frequencies of all the included variables in the model. The most missing were on the demographic and socioeconomic variables (2.9% – 2.6%) and were replaced by information from previous waves (t1-t4). There were less than 1.6% missing in the reset of the independent variables. Finally, we compared the results from the original data (without replacing missing data) with the results from the data were missing data were handled. Replacing missing data did not produce any substantial changes in the conclusions.

In Papers 2 and 3 we used strategies that are considered state of art when it comes to handling missing data. More specifically, in Paper 2 we used Multiple Imputation procedure (MI) in SPSS to deal with the potential bias arising from missing data in the predictor and confounder variables. The overall proportions of missing data among those who participated was low. In the NOR sample, the variable with the highest level of missing data was hyperactivity (4.3%), with proportions of missingness varying from 0.3% to 1.8% for the remaining variables. In the AUS sample, early sexual intercourse (26.4%) and mother’s education (7.1%) showed rather high proportions of missingness, whereas the remaining variables had between 0.3% and 2.5% missing data. Multiple imputation (MI) was used to handle missing data in the predictor and confounder variables, thereby providing missing data routines that are considered state of art and appropriate under missing at random (MAR) conditions (Schafer & Graham, 2002). As recommended, 20 complete datasets were created by imputation, incorporating all variables of interest (Schafer & Graham, 2002). All analysis and MI were conducted separately for the Norwegian and Australian sample. To test the robustness of our results, all analyses were additionally re-run by handling missing data by
listwise deletion. Finally, we compared the results from the original data (without imputation, using listwise deletion) with the results from the imputed data. MI did not produce any substantial changes in the conclusions. In Paper 3 we used The Full Information Maximum Likelihood (FIML) procedure in Mplus. Contrary to the MI procedure above, this procedure uses all available data in the analysis to estimate the model parameters without imputing missing data values (Graham, 2009).
4. Results

4.1 Paper 1:
Recent research suggests that early excessive drinking may be of greater importance for a wide range of subsequent adverse outcomes than early onset of drinking smaller amounts of alcohol. However, research on antecedents of early excessive drinking is scarce. Study 1 identifies predictors of early onset of intoxication (EOI) and whether they differ from those of early onset of drinking (EOD). The results showed that antecedents of EOI and EOD differ. Parental supervision, adolescents’ shyness, conduct problems and having friends who smoke regularly, use alcohol approximately once a week, have tried cannabis, or have been in contact with the police for illegal activities prospectively predicted EOI. Substantially fewer factors predicted EOD. When controlling for relevant covariates, EOI, but not EOD, were prospectively predicted by low levels of shyness, own conduct problems and having friends with deviant behaviour.

4.2 Paper 2:
Empirical studies have consistently shown that early onset of drinking is associated with alcohol-related problems in adulthood. However, recent reviews have identified several limitations in the early onset literature that question the nature of this association. This study addresses these limitations by examining whether early onset of drinking (EOD), independent of early onset of excessive drinking (EOE), prospectively predicts hazardous drinking in late adolescence/young adulthood in Norway and Australia, two countries with different drinking cultures. The results showed both EOD and EOE in adolescence to be related to an increased risk for alcohol-related problems in late adolescence/young adulthood, with a somewhat stronger association for EOE than EOD. Moreover, the associations were reduced, but remained statistically significant when controlling for a comprehensive number of potential confounders, including risk factors such as conduct problems, alcohol and drug use in the family and friends’ substance use. The same pattern of findings was identified for both the Norwegian and Australian samples – two countries with different alcohol policies and drinking cultures.

4.3 Paper 3:
Excessive alcohol use is common in adolescence and early adulthood and linked to injuries and other acute consequences. Less is known about the long-term outcomes of excessive drinking. This study examines how alcohol intoxication (AI) across adolescence to
young adulthood is associated with later levels of education, income and risk of
unemployment and disability. The results showed that early AI experiences (at age 13) was
significantly associated with higher risk of disability and unemployment and lower levels of
education and income at age 32. When controlling for covariates, the effects of AI at age 13
were markedly attenuated and most associations were no longer statistically significant.
By contrast, at age 22 and particularly age 31, the associations between AI and educational
and labour marked outcomes vanished or results even showed inverse associations.
Associations of AI with educational attainment and labour market integration at ages 22 and
31 remained similar when comparing unadjusted results with results with control for
covariates.
5. Discussion

5.1 Interpretation of main findings

The studies included in this thesis used longitudinal data to examine predictors of EOD and EOI and the prospective relationship between EOE/AI and important life outcomes, such as hazardous drinking, education, income and risk of unemployment and disability in young adulthood. Three main findings emerged. First, we found that predictors of EOD and EOI differed. All the included variables describing the adolescents’ own and friends’ norm-breaking behaviours were significantly related to EOI, but not to EOD. Second, contrary to our expectation, both EOD and EOE increased the risk of hazardous drinking in late adolescence/young adulthood, even after comprehensive adjustment for potential confounders. The pattern of results were furthermore similar across Norwegian and Australian samples. Third, early AI was related to increased risk of detrimental educational and work-related outcomes, but the relationship was confounded by the included control variables. AI in young adulthood represented no such risk. In the following, the main findings from the three studies comprising this thesis will be discussed in light of some of the theoretical concepts, proposed mechanisms and empirical findings described in the introduction.

5.1.1 Syndrome of problem behaviours

In the framework of PBT, adolescent alcohol use is not understood as an isolated behaviour, but rather as a part of a syndrome of problem behaviour. There is considerable evidence that young people’s substance use, including alcohol use, tend to co-occur with a wide range of other problem behaviours (Elliott, Huizinga, & Menard, 2012; Farrell, Danish, & Howard, 1992; Jessor, 1991). If early onset is part of a problem behaviour syndrome, we should be able to identify other risk factors in the behavioural system that predicts EOD. In the first study (Paper 1) we assessed conduct problems, covering a wide range of externalizing behaviours, and smoking as indicators of problem behaviours. Higher level of conduct problems and smoking were significant predictors of EOI, but surprisingly these factors seem to be unrelated to EOD. This contrasts with previous studies, where prior involvement in delinquent behaviour has been found to be one of the most consistent behavioural risk factors for staring to drink in adolescence (for a review, see Donovan, 2004). One possible explanation of why our findings differ from most of the literature may be that we are able to distinguish between those who have EOI and those who have EOD without having experienced intoxication, while most others define early onset of drinking to include both EOI and EOD. Our findings may thus indicate that EOI is more intertwined in the problem behaviour syndrome compared to
EOD. This may not be very surprising, and in Paper 1 we give a detailed account of possible explanations for this finding.

The finding that EOI to a greater extent than EOD seemed to be more intertwined in problem behaviours, was further supported by our finding that the most consistent predictor of EOI in our study, was having deviant friends. This factor was also unrelated to EOD in our study. The odds of EOI was almost two times higher for adolescents reporting having deviant friends compared to adolescents with no such friends after control for other variables (OR = 1.96, 95% CI: 1.14-3.35). Again, this is in contrast to previous empirical studies, where different aspects of peer deviancy (e.g., substance use, antisocial behaviour) are identified as key predictors of early onset of drinking (Donovan, 2004; Scholes-Balog et al., 2013; Trucco et al., 2011). In the PBT framework, peer deviancy is understood as an influence under the perceived environment system, with deviant peers functioning as models for and approving of, deviant behaviour. This implies a direct relationship between peer deviancy and early onset. An indirect function could be that substance-using peers may provide greater physical availability of alcohol. Nevertheless, the fact that the variable “deviant friends” in our study (Paper 1) predates EOI lends support to the notion that there is at least an element of social influence or peer pressure involved in the development of EOI. However, we cannot conclude that the involvement with deviant peers is the cause of EOI. There may be a selection process going on opening up for an interpretation of reciprocal causality. Adolescents may seek friends who are similar to themselves on certain characteristics. For example, adolescents willing to experiment with alcohol may be more likely to associate with other adolescents with the same willingness. This, in turn, increases the chances of early onset of intoxication.

In the framework of PBT, this common characteristic, which the selection process is based on, would perhaps be described as a general tendency towards unconventionality or proneness to problem behaviours.

In our study we identified lower levels of the temperamental trait shyness as a prospective predictor of EOI. Again, this trait differentiated between EOD and EOI, where lower levels of shyness significantly increased the risk of EOI but were unrelated to EOD. In our study (Paper 1), we argue that shyness may be seen in contrast to a tendencies to seek stimulating experiences, willingness to take risk and low levels of worry and rigidity, also captured in the concept of sensation seeking (Zuckerman, 1971). Such tendencies have been particularly examined in relation to drug use, (for a review, see Hittner & Swickert, 2006), but have also been identified as predictors of early onset (Webb et al., 1991). By contrast, shyness has been proposed to be part of a broader category of behavioural inhibition in both social and non-
social situations (Kagan, 2001). Consequently, we argue (in Paper 1) that the fact that lower levels of shyness differentiate between EOI and EOD, could indicate that shy adolescents may be more inclined to feeling anxious or withdrawing in social situations where alcohol is consumed and less inclined to engaging in heavy drinking, while lower levels of shyness may indicate a greater willingness to take risks.

In sum, the first study (Paper 1) demonstrated that, in contrast to previous studies, the adolescents’ own conduct problems, having deviant friends and having lower levels of shyness prospectively predicted EOI, but were unrelated to EOD. Based on these findings, it can be argued that EOI and not EOD, is part of the problem behaviour syndrome reflecting greater psychosocial unconventionality. The fact that previous studies have found associations between EOD and other non-conventional or norm-braking behaviours may imply that these studies have not distinguished between the two types of drinking behaviours and that it is EOI and not EOD that can be predicted by the above-mentioned factors. These findings raise the question of whether EOD and EOI relate differently to problem drinking in late adolescence/young adulthood. If it is in fact EOI that reflects greater social unconventionality, we would expect this behaviour to predict detrimental outcomes to a greater extent than EOD. This question was examined in the second study (Paper 2).

5.1.2 Early drinking behaviours indicates risk of hazardous drinking
The findings in the second study (Paper 2) showed that, contrary to our expectation, both EOD and EOE were related to an increased risk of hazardous drinking in late adolescence/young adulthood. Relative to abstainers, the prevalence ratio for hazardous drinking was approximately 2 times higher for adolescents reporting EOD and approximately 2-3 times higher for adolescents reporting EOE. This relationship was observed even after comprehensive adjustment for potential confounders, and the pattern was similar in the Norwegian and Australian sample. Based on these findings, it seems that early adolescent alcohol use, regardless of the level consumed, is a robust indicator of risk of subsequent alcohol-related problems.

Lending support from the PBT framework, this association would be explained in light of the concept of psychosocial proneness to problem behaviours, where EOD and EOI, as well as adult problem drinking are all expressions of such proneness. The results in Paper 1, however, suggests that this cannot fully explain the observed relationship, as adolescents with EOD does not seem to show characteristics indicating greater proneness to problem behaviours. An alternative explanation, lending support from the theories of compromised
biological and cognitive development (DeWit et al., 2000; Guttmannova et al., 2011) or compromised social, emotional and cognitive development (Jessor, 1991; Pedersen & Skrondal, 1998) described in the introduction, would be that early alcohol use, either biologically or socially, changes the course of development leading to adverse adult outcomes. However, as pointed out by others (e.g., Rossow, 2006), it is difficult to see why having more than just a few sips, as is how we have operationalized EOD, should have such profound impact on adolescent development. One could assume that the likelihood of such changes taking place would be greater when the adolescents consume alcohol to the level of getting drunk, presuming that this may have a toxic effect with the potential to invoke neurobiological changes in the brain. Furthermore, getting drunk at a young age is presumably a greater norm transgressing behaviour, and as such has more potential to change the identity or role, as compared to having a few sips in for example a family dinner. However, the findings in the second study (Paper 2) show only minor differences in how EOD versus EOE prospectively predicts later hazardous drinking behaviour, and the difference was not statistically significant. More importantly, the theories of compromised social and biological development, would be more plausible if the relationship between EOD and alcohol-related problems more consistently demonstrated a robust association across different studies in the field. As described in the introduction, there are still some contrasting results as far as the prospective relationship between early onset and adult alcohol related outcomes is concerned. Some studies demonstrate that the associations are completely explained by relevant covariates (King & Chassin, 2007; Newton-Howes & Boden, 2016; Rossoow & Kuntsche, 2013), while other prospective studies do not support this pattern of findings (Buchmann et al., 2009; Irons et al., 2015). Thus, it seems that neither the psychosocial proneness theory nor the theory of disturbed social or biological development serve as satisfactory explanatory models for the findings in our studies.

Our findings in the second study (Paper 2) are in line with the findings of Buchmann et al. (2009) and Irons et al. (2015). When contrasting these studies and ours to those studies where the association was completely explained by the included covariates (e.g., conduct problems, parental drinking patterns) (King & Chassin, 2007; Newton-Howes & Boden, 2016; Rossoow & Kuntsche, 2013), it became evident that the age at which adult alcohol-related drinking problems were assessed, differed. In Paper 2, we thus argue that the contrasting findings may be understood in light of what life phase the young adult drinking is measured and what heavy drinking signifies in different adult life phases. Our study, as well as the studies in line with our findings, have assessed problem drinking in the late
adolescent/young adult period (ages 18 – 25), while the studies with contrasting findings assess problem drinking in the young adult/adult period (ages 25 and above). The late adolescent/young adult period has been characterized as the “prime drinking years”. During this life phase, excessive drinking is highly prevalent and accepted (Schulenberg & Maggs, 2002). Continuance of a hazardous drinking pattern in the young adult/adult period, on the other hand, where obligations to work, partner and perhaps a family are greater, may have different connotations. In particular, individuals who maintain an excessive drinking pattern, when most people mature out of this drinking pattern when facing adult obligations, are characterized by more severe and long-lasting problems (Schulenberg & Maggs, 2002). We suggest (Paper 2) that what hazardous drinking may reflect in these different life phases, a socially accepted behaviour versus a behaviour with the potential of greater social costs, may provide important information about the nature of the contrasting findings described above.

More specifically, socially accepted excessive drinking may to a larger degree be based on social and environmental factors, including early alcohol exposure, than maintenance of such a high-risk drinking pattern into the adult life phase. A hazardous drinking pattern in this period of life may be better understood in the framework of underlying genetics. This may explain why studies of the relationship between EOD/EOE and hazardous drinking in the adult life phase find that the relationship is confounded by indications of more complex problems, such as conduct problems or parental drinking, variables thought to reflect genetic transmission or vulnerability. As such, these studies add support to the notion of early onset being a general marker of risk rather than an independent risk factor. Our findings on the other hand, where the relationship between EOD/EOE and hazardous drinking in the late adolescent/young adult period was not confounded by the same variables, could be taken as support of the proposed causal explanations. Our study design is however unfit to rule out all other plausible explanations for the effect, and as such cannot identify a causal mechanism (cf., Shadish et al., 2002). Instead, we suggest that the results from the second study (Paper 2) support the idea that associations between early drinking and later hazardous drinking vary according to the age at which drinking outcomes are assessed. We furthermore suggest that the influence of EOD and EOE may be relatively short term.

In sum, our study adds to the literature by demonstrating that early drinking behaviour, regardless of level, is an indicator of alcohol-related problems in late adolescence/young adulthood. However, the divergence in findings across prospective studies of younger versus older adults, may suggest that the predictive power of EOD/EOE for alcohol-related problems is limited to the late adolescent and young adult period.
5.1.3 From a marker of risk to a marker of social integration

Our findings in the third study (Paper 3) may provide important information concerning the potential role of early alcohol intoxication (AI) as a risk factor for detrimental educational and occupational outcomes. The results in Paper 3 showed that AI at age 13 was significantly related to lower levels of income, higher risk of unemployment and lower educational attainment later in life. However, most of the associations disappeared when controlling for relevant covariates, such as conduct problems, mental health, grades in school, parents’ alcohol use and parents’ education level. All these factors tap into individual and familial genetic and environmental risk, as described in the introduction. These findings add support to empirical studies suggesting that early alcohol use may be part of a broader set of problem behaviours (e.g., Rossow & Kuntsche, 2013), or a manifestation of an underlying vulnerability (e.g., King & Chassin, 2007; Ystrom et al., 2014), rather than an independent risk factor per se. As such, early AI can be understood as a marker of risk, not only for alcohol-related outcomes in adulthood, as most previous empirical studies have demonstrated, but also in relation to educational attainment and labour market integration.

Interestingly, in our study (Paper 3), AI in early and middle adulthood did not function as a marker of risk, rather the opposite. AI in the early twenties (age 22) and early thirties (age 31) was in fact related to higher income and partly lower risk of disability compared to those without AI. Furthermore, these associations were not attenuated by the included covariates. There may be several explanations for this pattern of findings. As described in the introduction, although it has been suggested that moderate drinking improves individual’s health and hence productivity and earnings (Barrett, 2002), there is little empirical evidence to support a claim that AI causes educational and work-related success. Rather, as our study demonstrated striking age differences in how AI was related to future educational and labour marked outcomes, we suggest that what alcohol use, and particularly heavy use, signifies in different phases of life, provides important information when interpreting the findings.

More specifically, at age 13, most adolescents do not engage in AI (Kraus & Nociar, 2016). Getting drunk at this early age is, in most western countries, a clear breach of socially accepted behaviour. Getting drunk in the early twenties and thirties, however, is more socially accepted, at least in Norway (Nordlund & Østhus, 2013). More importantly, drinking alcohol, even excessively, is a central part of social gatherings in both private and work-related settings in adulthood. The same cannot be said about the role of alcohol in early adolescence, where only 4% of adolescent intoxication (aged 14-17) episodes takes place in social events at
school or in connection with organized leisure activities as the location for their drinking (Storvoll, Rossow, & Pape, 2010). As such, we argue (in Paper 3) that as most adolescents do not engage in intoxication drinking in early adolescence, AI can be understood as a norm-transgressive behaviour and a part of problem behaviour, as described in the PBT framework. AI in early and middle adulthood, on the other hand, can be understood as not only a normative and highly prevalent behaviour, but also a behaviour that reflects social integration, such as having close friends, having a large social network, being popular and having an active social life. Understood this way, it does not come as a surprise that AI in early and middle adulthood is positively related to work-related outcomes, such as income. In these age groups, AI may be understood as a marker of social integration.

The understanding of early adolescent AI as a non-normative behaviour intertwined in problem behaviour, and early and middle adult AI as a normative behaviour intertwined in prosocial behaviour, is furthermore supported by our findings that the relationship between early adolescent AI and work related outcomes was confounded by factors such as conduct problems, school grades and parental background, while the same was not the case for AI in early and middle adulthood. The associations between AI in early and middle adulthood and positive work-related outcomes are more likely to be confounded by different variables reflecting well-adjustment. It is however important to note that in the third study (Paper 3) we have most likely not been able to identify true high-risk user groups in young adulthood, as being intoxicated more than 10 times the last 12 month (the highest cut-off chosen in the study) may be too low to identify alcohol users with a clearly problematic pattern of AI. Unfortunately, the number of participants with higher frequency of AI (i.e., > 50 the last 12 month) was too small to allow for differentiation of more excessive AI at all ages. As such, had we been able to differentiate a high-risk group as well, the relationship between AI in early and middle adulthood and work-related outcomes may have looked differently.

In sum, the third study (Paper 3) contribute to the understanding of the relationship between AI across adolescence and educational attainment and labour marked integration in adulthood and provide evidence that the association is of a non-causal nature. Furthermore, the results show that the role of AI changes during the developmental course from a marker of risk to a marker of social integration.
5.2 Methodological considerations

The studies presented in this thesis have several strengths and address several of the limitations previously identified in the literature of predictors of early onset and its associations to subsequent outcomes. First, the use of prospective design from childhood to young adulthood allowed us to identify predictors of EOD and EOI that are present before the drinking behaviour starts. Second, “onset” was measured close in time to when the adolescents start to drink, minimizing the potential bias caused by retrospective recall or forward telescoping as often noted as a major limitation in the literature on early onset of alcohol use (Rossow, 2006). Third, the comprehensive set of variables included from different domains of influence enabled an evaluation of a comprehensive set of risk factor for different types of drinking behaviour, and a rigorous control of potential confounders. Fourth, the consideration of the different early drinking behaviours and the use of two samples from different countries with different prevention policies and drinking cultures, makes an important contribution to the literature. Finally, the long follow-up period from early adolescence to young adulthood, and linkage to national registers, enabled an elucidation of age-specific aspects of intoxication drinking in relation to long-term outcomes. Taken together, these aspects make the studies comprising this thesis important contributions to the literature on predictors of early onset and its association to important life outcomes. There are, however, several limitations in these studies that must be acknowledged and discussed, which will be done in the following sections.

5.2.1 Construct validity and reliability

In the current project, we use adolescent and young adult self-report of alcohol use. There is a considerable amount of research supporting the validity and reliability of adolescents’ self-report measures on alcohol use and problem drinking (Barnea, Rahav, & Teichman, 1987; Lintonen & Rimpelä, 2001; O'Callaghan & Callan, 1992; O’malley, Bachman, & Johnston, 1983; Torsheim, Wold, Samdal, & Haugland, 1997). However, some issues regarding possible shortcomings using questionnaires, which may cause unreliability in adolescent drinking measurement, needs to be considered. For example, in the alcohol literature it has been suggested that there is a tendency to systematically under-report alcohol use, but that this tendency may be more pronounced among adults (and especially the moderate and high consumption groups) than among adolescents (Lintonen, Ahlström, & Metso, 2004). The under-reporting is often understood in light of the social desirability theory - the desire to put oneself in a favourable light (see e.g., Davis, Thake, & Vilhena, 2010). This desire in relation
to alcohol use may be more important for adults, who in their own opinion and perhaps in the public eye, drink to a level that is not socially acceptable. In adolescence, however, experimentation with alcohol in mid-adolescence is to some extent normative in the peer group. In this way, alcohol use is not considered undesirable and may thus not represent a barrier to reporting use (Lintonen et al., 2004).

On the other hand, one could suspect that over-reporting would be a greater risk in this age group, as adolescents tend to exaggerate the drinking of other adolescents, creating an imaginary group pressure, a phenomenon known as the majority fallacy (Bruun & Hague, 1963). Another, perhaps somewhat related phenomenon, is “faking bad” (Babor, Brown, & del Boca, 1990), i.e. intentionally over-reporting drug use (and other risk-taking behaviours) out of a desire to give a negative impression of oneself. In the current study, this may have resulted in a slight over-reporting of drinking experiences in the youngest age group, and a slight underreporting in the young adult group. Our measures of EOD/EOI/EOE/AI gives us no indication of the actual volume of alcohol consumed, and an objective measure of alcohol consumption and drinking patterns would have been an advantage in addition to knowledge of drinking context. However, the better differentiation of early drinking experiences into EOD and EOI/EOE improves upon previous studies.

The concept of “Hazardous drinking” that we use in Paper 2 is based on a well-validated screening instrument; the Alcohol Use Disorders Identification Test (AUDIT) (Babor et al., 2001). Although this instrument is validated in different countries, it is not necessarily correct to assume that this instrument captures the same phenomena across the Norwegian and Australian samples. In particular, the "young adult" age range is different between the two samples (Norwegian: 18.3 – 19.9 years, Australian: 22.9 – 24.6). This is an age of many transitions, and for the Norwegian sample, this includes a recent transition to legal drinking age, while the Australian sample has had the opportunity to purchase alcohol for several years and as such may have more experience drinking. This might impact results, as the Australians may be in a slightly different life phase, somewhat more “settled down” compared to the Norwegian adolescents, who just reached legal drinking age and are just at the verge of entering adult roles and responsibilities. This might be reflected in the different rates of hazardous drinking, which is higher in the Norwegian sample compared to the Australian. Another point is that we have examined the relationship between variables, with a main focus on EOD, EOI and Hazardous drinking, in two Western countries. Although we argue that the pattern of relationship is similar across the cultural contexts, based on the different prevention
policies and drinking cultures, we could have considered including other, non-western countries.

In all three papers, we use several scales as predictors and covariates. The Cronbach’s alphas reported, were generally higher than .70, indicating acceptable internal consistency. A few were below .70, indicating questionable internal consistency (i.e., conduct problems in the Australian sample in Paper 2: $\alpha = .64$, and the parenting scales inconsistent discipline: $\alpha = .67$ and other disciplinary practises: $\alpha = .65$ in Paper 1). One scale, the impulsivity scale used in Paper 2, displayed poor internal consistency ($\alpha = .57$). The Cronbach’s alpha value is, however, sensitive to the number of items in the scale, and in short scales (e.g., scales with fewer than ten items), such as the conduct problems scale (9 items), parenting scales (4 and 7 items respectively) and the impulsivity scale (3 items), it is common to find quite low Cronbach values. The number of items could thus explain the moderate alphas. Still, the role of these measures in predicting adolescent drinking behaviours should be interpreted with caution.

Another concern is related to how we measured substance use or alcohol problems in the family/household. These measures are included because of the strong empirical support of the relationship between indicators of family alcohol problems and early initiation and alcohol-related problems in young adulthood. However, we may not have captured this phenomenon in a satisfactory manner in our studies. In Paper 1, alcohol problems in the household were defined to have occurred if the mothers reported “alcohol problems in the household” one or more times the past 12 months at t1, t2, t3, t4 or t5. As such, we do not know exactly who the problem refers to, nor the permanence of the problem in the household (e.g., a substance-using partner may have been a part of the family for a short or a long period). The same concern goes for Paper 2, as we here asked the adolescents to consider the statement “one of the people closest to me uses too much alcohol, pills or other drugs” in the Norwegian sample, and ”Has anyone in your family ever had a severe alcohol or drug problem” in the Australian sample. It is unclear which member of the family these statements refer to, whether it is a member of the household or a more distant relative, and in the Norwegian sample, it is unclear whether the substance use can be considered a substance use problem.

5.2.2 Representativeness and generalizability
Characteristics of the sample and patterns of attrition are aspects which may have implications for the representativeness and generalizability of the findings presented in this thesis. In the following section, some of these aspects are accounted for and discussed.
The sampling procedure and initial response rate is important to consider in relation to representativeness. In the TOPP study, 84% (n = 913) of the invited families (n = 1,081) participated with information about their children. Based on comparisons of the participating mothers and the non-responding mothers in the initial TOPP sample (Mathiesen & Tambs, 1999), and comparisons of the initial TOPP families with 1,000 families from a sample using healthcare centres in all of Norway (Sætre, Mathiesen, & Nærde, 1996), the TOPP sample has been considered reasonably comparable to the Norwegian population of families with small children on sociodemographic variables and family stressors. However, the sample is comprised of mainly ethnic Norwegian families, limiting the generalizability of the findings to other ethnic groups, in Norway as well as in other countries, with different attitudes towards alcohol use. The IYDS recruited a total of 165 classes in 152 schools, which constituted 65% of the eligible classes selected in Victoria. The participating schools were compared to overall state statistics on three school-level indicators: school type, economic disadvantage and student diversity. The school samples were found to be a good representation of the school-aged population of Victoria, Australia (McMorris et al., 2007). In all, 3,926 students were eligible for participation, of whom 2,884 (74%) consented and participated. The initial YiN sample included students in grades 7 through 12 (ages 12 to 20, N = 12, 287) from 67 schools representative of high schools in Norway, with a response rate of 97% (Wichstrøm & Wichstrøm, 2009). Every school in the country was included in the register from which schools that participated were selected. In Norway, 98.5% of the age cohorts between 13 and 16 attend the ordinary public junior high schools. As such, the sample was drawn from a population comprising almost the entire age cohort. The sample was furthermore stratified according to geographic region and school size, which in Norway is closely related to the degree of urbanization (von Soest et al., 2016). The sampling procedure and high response rate in the YiN study, resulted in a sample with good representativeness of the adolescent student population in Norway.

Although the initial response rates were high (84% in TOPP, 76% in IYDS and 97% in YiN), both TOPP and YiN had substantial drop-out rates over time. At the final data collection, 47% of the children participating at t1 in TOPP responded to the questionnaires. Of those eligible for follow up in YiN, 66.9% responded at t4. It is not uncommon to have attrition rates as high as 40 to 60% in longitudinal studies (van der Kamp & Bijleveld, 1998). It is, however, the degree to which the drop-out is systematic that might represent a problem for the generalizability of the findings to the general population. As described in the methods section, the only systematic difference between those adolescents who dropped out and those...
who remained was in the TOPP study related to lower level of maternal education and being male (Gustavson et al., 2012; Kjeldsen et al., 2016). In YiN (see also methods section), attrition was predicted by being male and low parental socioeconomic (Wichstrøm, 2000a, 2000b; Wichstrøm & Hegna, 2003). This may represent a threat to the representativeness and findings may be somewhat more uncertain in terms of people with low socio-economic status (Papers 1-3). Attrition was also related to few hours spent on homework, poor school grades and conduct problems in the YiN study (Wichstrøm, 2000a, 2000b; Wichstrøm & Hegna, 2003), resulting in an overrepresentation of adolescents who spend more time on homework, receive superior school grades and have lower levels of conduct problems. This selective attrition may to a certain degree influence estimates of the relationship between AI and outcome variables (Paper 3). However, a Monte Carlo simulation has demonstrated that estimates of associations between variables tend to be more robust against selective attrition than estimates of means and frequencies (Gustavson & Borren, 2014). This indicates that valuable knowledge can be gained of the relationship between variables despite the presence of selective attrition.

A final aspect relates to whether our findings can be generalized to other countries and cultural contexts. Norway is characterized by a one of Europe’s strictest alcohol regulation policies (Österberg & Karlsson, 2003), overall low levels of alcohol consumption compared to most European countries (Babor, 2010), a drinking pattern characterized by excessive drinking at weekends (Nordlund & Østhus, 2013) and well-developed welfare schemes aimed at fighting social exclusion, as well as low prevalence of unemployment. Such characteristics may limit generalizability to other countries with different national policies and cultural characteristics. For instance, the predictive power of early adolescent alcohol use on adult outcomes may depend on cultural factors, with research showing that in permissive cultures where moderate drinking among adolescents is culturally normative, EOD is to a lesser degree linked to other markers of deviance, such as conduct problems, than in cultures were EOD is considered a greater social problem, such as the United States, where most of the research on early onset has been conducted (Geels et al., 2013). More specifically, adolescent drinking behaviours take on meaning in the larger cultural and political context, and as such may relate differently to adult outcomes depending on what that particular behaviour signifies in a specific context. Still, we found similar pattern of findings in Norway and Australia, two western countries with different alcohol policies and drinking cultures (Paper 2). This may indicate that the findings may be generalizable to at least Western countries.
5.2.3 Multiple significance testing and small sample size

A concern in Paper 1 and 2 is that we have many predictor and control variables in the models (20 and 14 respectively), which might increase the risk of type I error (false positives). Running multiple significance test increases the risk of getting significant results by chance alone. Adjusting for the number of tests, could reduce this risk, but also reduces statistical power, which in turn, especially with a relatively small sample size that we have in this two studies, increases the risk of type II error (false negatives).

Statistical power also needs to be considered in relation to the sample size, as it may affect the ability to detect effects that actually exist (Shadish et al., 2002). For example, in Paper 1, our analytical strategy was to include only significant variables from the simple analyses (adjusting only for age, gender and alcohol use before 12.5) in the multivariate analysis. There is a possibility that a larger sample size would have revealed that more variables were related to EOD and EOI/EOE (e.g., alcohol problems in the household). Furthermore, there were only 43 adolescents who reported EOI. However, in spite of the relatively low number of adolescents in this group, several significant associations between predictors and EOI were found, indicating that the effects that were obtained in the analyses were in fact of considerable size.

The third study (Paper 3) was based on a larger sample (n=2,602). This large sample size ensures high statistical power, thus reducing the risk or Type II error, i.e., obtaining non-significant results for associations between variables that actually do exist in the population. In Paper 3, relationships over a great time span (19 years) to outcomes that are not prevalent during young adulthood (i.e. unemployment and disabled or on rehabilitation) were examined, and significant long-term associations between adolescent drinking behaviours at age 13 and all life-outcomes in young adulthood were found, suggesting that we had high statistical power to detect effects.

5.3 Concluding remarks and implications

This thesis add support and extend previous research in several ways. First, the studies included in this thesis address several limitations identified in reviews of the early onset literature by: (a) identifying antecedents of early onset of intoxication and whether they differ from those of early onset of drinking, (b) providing information about whether having consumed small amounts of alcohol at an early age is related to adverse alcohol-related outcomes, or whether such associations are first seen when more substantial amounts are consumed, (c) using a cross-national design to examine whether associations between early
onset and adverse alcohol-related outcomes vary across cultural contexts, (d) examining age-specific aspects of alcohol intoxication in relation to important life outcomes other than drinking pattern, (e) examining early onset and associations with future outcomes prospectively and (f) including a wide range of possible confounders. This has resulted in a better understanding of the role of early drinking experiences and short- and long-term associations to adult outcomes.

In particular, our findings show that early onset of intoxication is predicted by variables reflecting the adolescents’ own and their friends’ norm-breaking behaviours, whereas such factors were unrelated to drinking smaller amounts of alcohol (Paper 1), but that both drinking behaviours, regardless of level, are indicators of alcohol-related problems in late adolescence/young adulthood (Paper 2). This pattern is consistent across samples drawn from two countries with different prevention policies and drinking cultures. These findings are in contrast to previous research on older adults, where no association between adolescent drinking and later alcohol problems was found when controlling for covariates. The divergence in findings across prospective studies of younger versus older adults, may suggest that ability of EOD/EOE to predict alcohol-related problems is limited to the late adolescent and young adult period. Also of importance is our contribution to the understanding of the relationship between alcohol intoxication across adolescence and educational attainment and labour marked integration in adulthood. Our findings provide evidence that the association is of a non-causal nature and furthermore, that in early teenage years, alcohol intoxication was a predictor of subsequent poor educational and labour market outcomes. However, from early 20s onwards, measures of AI changed character and rather became a predictor of good outcomes in several labour market-related domains.

Although the studies included in this thesis identifies several risk factors or prospective predictors of problem development, this does not automatically indicate effective prevention interventions. This would require that the causal relationship between the variables were identified, or that the mechanism explaining the associations was fully understood. Our studies do not provide such information. Still, the findings are valuable in that they provide indicators of at-risk groups, and as such allow for a better targeting for preventive and early intervention efforts.

In particular, although we have no firm basis to suggest that a delay of drinking onset or onset of excessive drinking would prevent the overall development of adult alcohol problems, our findings nonetheless indicate that efforts to reduce early age drinking and reduce the progression from experimental/minor alcohol use to instances of excessive
drinking may have some impact on the risk of alcohol-related problems in the late adolescence/young adulthood period. The impact may, however, be limited to the late adolescent and young adult period. Although we do not know the exact nature of the relationship between early drinking behaviours and adult outcomes, preventing early drunkenness is an important goal in itself, as immediate consequences of excessive alcohol use, such as alcohol-related accidents, can be avoided. Our identified predictors of EOI indicate that prevention strategies may benefit from including non-alcohol-specific prevention efforts implemented in late childhood/early adolescence targeting factors in multiple domains, including family and peers. Moreover, interventions aimed at enhancing young people educational attainment and labour marked integration in adulthood may very well also benefit from targeting early intoxication drinkers. However, the efforts are not likely to have impact if not tackling a wider range of familial and individual vulnerability factors.

The findings in this thesis also provide some direction for future research. In particular, future longitudinal studies should examine the role of early drinking behaviours and indicators of alcohol-related and work-related problems in different life phases and in different cultural contexts. Furthermore, more research is needed to shed light on the potential mechanisms in which early drinking behaviours are prospectively associated with detrimental life outcomes in adulthood and in which heavy alcohol use from the early 20s is prospectively related with good labour market outcomes. Such studies could inform health promotion, prevention and early intervention policies in important ways.
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Papers I-III
Predicting early onset of intoxication versus drinking—A population-based prospective study of Norwegian adolescents

Frøydis Enstad⁎, Willy Pedersen, Wendy Nilsen, Tilmann von Soest

Aims: Recent research suggests that early onset of intoxication (EOI) may be of greater importance for a wide range of subsequent adverse outcomes than early drinking experiences without intoxication. However, research on antecedents of EOI is scarce. The present study identifies predictors of EOI and whether they differ from those of early onset of drinking (EOD).

Methods: Data was drawn from the prospective Tracking Opportunities and Problems (TOPP) study of Norwegian families (n = 382), which followed up mothers and their children with six data collections from childhood (age 1.5) to adolescence (age 14.5). Self-reports from the adolescents (parenting practices, adolescent's conduct problems and friends' deviant behaviour) and their mothers (adolescent temperament, socio-economic factors and household alcohol problems) were used to identify predictors of EOI and EOD.

Findings: A variety of temperamental, socio-economic, and family factors predicted EOI, whereas EOD was predicted of substantially fewer variables. Particularly, when controlling for relevant covariates, low levels of shyness, own conduct problems and having friends with deviant behaviour prospectively predicted EOI, but not EOD.

Conclusion: Future research and prevention efforts should take into consideration that EOI and EOD without getting drunk appear to be predicted by different risk factors in childhood and adolescence.
have examined whether such predictors differ from those of EOD. We were able to identify only one longitudinal study focusing explicitly on whether potential risk factors predicted different early drinking behaviours differently (onset drinking, onset drunk and onset binge drinking) (Jester et al., 2015). This study reported that higher expectancies for alcohol use and norms regarding “drunken comportment” vary between different cultures (MacAndrew & Edgerton, 1969). The majorities of studies on EOD are carried out in the US. Thus, there is a need to examine whether the risk profiles for both EOD and EOI identified in the literature also hold in different cultural contexts with varying alcohol-related drinking patterns and norms. The current study is set in the Norwegian cultural context, which is characterized by a strict alcohol regulation policy, influenced historically by a strong temperance movement, and somewhat paradoxically, a drinking culture characterized by excessive drinking at weekends. The primary aim of this study is to examine predictors of EOI in the age span from early childhood (1.5 years) to middle adolescence (14.5 years) using multi-informant information. We will also examine whether such predictors differ from those for EOD without EOI. The models include a wide range of prospective parent and adolescent self-reported risk factors (i.e., temperament, socio-economic factors, household alcohol problems, parenting practices, adolescent smoking, drinking and conduct problems and friends deviant behaviour) that have previously been associated with EOD, simultaneously in models predicting EOD and EOI relative to abstainers and relative to each other.

2. Methods

2.1. Participants and procedure

The sample was drawn from the Norwegian population-based prospective study Tracking Opportunities and Problems (TOPP) where mothers and their children are followed over an 18-year span. Originally 1081 families from 19 geographical health care districts in eastern Norway (28% living in large cities, 55% in densely populated areas and 17% in rural areas), were invited to the study. They were recruited when the families attended their toddlers’ 18 month vaccination in 1993 at the child health clinics. Details of the study are described elsewhere (Nilsen et al., 2017; Mathiesen, Tambs, & Dalgaard, 1999). The participants have been followed up over eight data collections from when the children were 1.5 years (T1) to 18.5 years (T8). Questionnaires were handed out and returned at the clinic in the three first waves. The remaining surveys were conducted by mail. From age 12.5 (T5) and thereafter, the adolescents replied to their own questionnaire. At T1, 85% (n = 913) of the invited mothers participated. Background data from the child health clinics at 1.5 years showed that non-respondent mothers did not differ significantly from responding mothers in age, education, employment status, or marital status (Mathiesen & Tambs, 1999). Attrition over time was predicted by lower educational level at baseline (Gustavson, von Soest, Karevold, & Roysamb, 2012). The current sample includes self-report data from the mothers of children aged 1.5 to 14.5 (response rate: 51.9%, calculated on the basis of participation at T1), and adolescents at 12.5 (T7, response rate: 61.9%) and 14.5 years (T6, response rate: 50.2%). In all, adolescent and mother reported data from 382 participants were available and comprised the current sample. The participants gave their informed consent and the study was approved by the Regional Committees for Medical and Health Research Ethics.
3. Measures

3.1. Alcohol use

At 12.5 and 14.5 years alcohol use and intoxication were measured by adolescent self-report on two items: “Have you ever tasted more than a few sips of alcohol?” and “During the past 12 months, have you had so much to drink that you felt clearly intoxicated?” with five response categories (Never, Once, 2–5 times, 6–10 times and > 10 times). These items were categorized into three groups at 14.5 years: 1) Abstinent (never tasted, never been drunk), 2) EOD (tasted one or more times, but never been drunk) and 3) EOI (tasted one or more times and been drunk one or more times).

3.2. Temperament

Adolescent temperament was assessed at age 12.5 by maternal report on the EAS Temperament Survey for Children; Parental ratings (Buss & Plomin, 1984). The scale assesses the adolescents’ degree of emotionality (the tendency to become aroused easily and intensely, 12 items), activity (preferred levels of activity and speed of action, 4 items), sociability (the tendency to prefer the presence of others to being alone, 4 items) and shyness (the tendency to be inhibited and wary in new social situations, 4 items). Responses were rated on a 5-point scale ranging from 1 (Not typical) to 5 (Very typical). Mean scores were computed and Cronbach's alpha at age 12.5 for the four temperamental subscales were 0.81 (emotionality), 0.82 (activity), 0.63 (sociability) and 0.77 (shyness).

3.3. Socio-economic factors

Family status was reported by the adolescents at age 12.5 and recoded into a dummy variable where those living with both biological parents (value 1) were contrasted with all other living arrangements (value 2). Maternal education was assessed by asking the mothers to report their highest level of education on a scale from 1 (9 years primary school or less) to 5 (> 4 years at university or university college). Employment measures the mothers’ workforce participation in terms of percentage of paid work (1 = No paid work, 2 = < 50%, 3 = 50–80% and 4 = 80–100%). Household economy was measured by asking the mothers “How do you/your family cope with your current financial situation?” with five response categories ranging from 1 “We cope very well” to 5 “We cope very poorly”.

3.4. Family factors

Alcohol problems in the household were defined to have occurred if the mothers reported having experienced “alcohol problems in the household” one or more times during the past 12 months with children aged between 1.5 and 12.5 (0 = No, 1 = Yes). Parenting practices at age 12.5 were measured by the Alabama Parenting Questionnaire (Shelton, Frick, & Wootton, 1996), which measures positive involvement with children (Cronbach’s alpha 0.76), use of positive discipline techniques (Cronbach’s alpha 0.70), consistency in the use of such discipline (Cronbach’s alpha 0.67) and other disciplinary practices (Cronbach’s alpha 0.65). Items were rated on a 5-point Likert scale from 1 (Never) to 5 (Always) and mean scores were computed. We also included a 10-item revised subscale on parental strictness and supervision from the Lamborn Parenting Scales (Lamborn, Mounts, Steinberg, & Dornbusch, 1991) at age 12.5, with response categories recoded into “Low” (coded 1) to “High” (coded 3) levels of strictness/supervision. Mean scores were computed and Cronbach alpha was 0.75.

3.5. Adolescents’ and friends’ behaviour

Adolescent smoking was assessed by one item with responses ranging from 1 (“Have never smoked”) to 4 (“Smoke daily”) at age 12.5. Adolescent conduct problems were assessed by 22 items with response options ranging from 1 “Never happened” to 5 “Happened > 10 times”. The scale is based on three different Scandinavian scales of antisocial behaviour and is described in more detail elsewhere (Kjeldsen, Janson, Stoolmiller, Torgeren, & Mathiesen, 2014). Deviant behaviour of friends was assessed by four items asking about number of close friends who smoke regularly, use alcohol approximately once a week, have tried cannabis, or have been in contact with the police for illegal activities. The response categories were 1 (none), 2 (one friend) and 3 (several friends). Mean scores were computed for both scales.

3.6. Covariates

Gender, age and alcohol use before age 12.5 were used as covariates in most analyses.

3.7. Statistical analyses

Predictors of EOI and EOD at age 14.5 were examined by means of multinomial logistic regression analyses. The variables in the models were categorized under four domains: temperament, socio-economic factors, family factors and adolescents’ and friends’ behaviour. Firstly, all variables were evaluated one by one in a series of multinomial logistic regression analyses, with control for age, gender and alcohol use before age 12.5. Being abstinent was chosen as the comparison group; thus each analysis produced two comparisons: the odds of EOI and EOD compared to abstinent. In order to identify predictors that differentiate between EOI and EOD, we conducted additional analyses using EOD as comparison group. Secondly, we entered all significant predictors from these analyses simultaneously into multiple multinomial regression analyses. All models were adjusted for age, gender and alcohol use before age 12.5. Participants with intoxication experiences before the age of 12.5 were excluded from the analysis in order to predict EOD and EOI by age 14.5 without including respondents who had been drunk before this age.

4. Results

Prevalence of alcohol use is provided in Table 1. Adolescents reporting alcohol use increased from 16.4% to 38.9% from age 12.5 to 14.5, with the majority (82.6% and 61.3%) being abstinent in both age groups. Four participants (1.0%) reported having been drunk before age 12.5. Moreover, 105 (27.5%) respondents reported EOD (without having been intoxicated) at 14.5 years.

Results from multinomial logistic regression analyses examining the relationship between each predictor of EOI and EOD separately, with control for age, gender and alcohol use before age 12.5, are presented in Table 2. When comparing adolescents who reported EOI with those who had remained abstinent, significant relationships between predictors and outcome were revealed in all four domains. In the temperamental domain, low levels of shyness and high levels of sociability significantly predicted increased odds for EOI. Of socio-

### Table 1

<table>
<thead>
<tr>
<th>T5 Age 12.5</th>
<th>T6 Age 14.5</th>
</tr>
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<tbody>
<tr>
<td>Intoxication/EOI</td>
<td>1.5% (48)</td>
</tr>
<tr>
<td>Alcohol use/EOI (without intoxication)</td>
<td>16.4% (63)</td>
</tr>
<tr>
<td>Abstinent</td>
<td>82.6% (319)</td>
</tr>
<tr>
<td>Total</td>
<td>100% (386)</td>
</tr>
</tbody>
</table>

* The 4 respondents reporting having been intoxicated at age 12.5 are excluded from all further analyses.
economic factors, only better household economy was related to a decreased risk of EOI. In the family factors domain, a parenting style characterized by greater involvement and higher levels of strictness and monitoring significantly reduced the odds of EOI compared to abstinent. Finally, all predictors concerning the respondents’ and their friends’ behaviour was significantly related to EOI, with high levels of problem behaviour and more deviant friends being associated with increasing risk of EOI.

When comparing EOD to abstinent, comparatively fewer variables significantly predicted the outcome, as only high levels of emotionality, low household economy, alcohol use before age 12.5, and female gender were significantly related to increased risk for EOD at age 14.5. None of the predictors within the family factors and adolescents’ and friends’ behaviour domain were significantly related to EOD.

Finally, when predicting EOI compared to EOD, adolescent showed a higher risk of EOI when reporting low levels of shyness, high levels of conduct problems, and having friends with deviant behaviour.

Next, multiple multinomial logistic regression analyses were conducted, where all significant predictors from previous analyses were included simultaneously in one model (see Table 3). When predicting EOI compared to abstinent, low levels of shyness and parental strictness and supervision remained significantly related to EOI. Likewise, conduct problems and deviant friends remained significant predictors, and female gender and alcohol use before age 12.5 significantly increased the odds of EOI.

When comparing EOD to abstinent, in addition to alcohol use before age 12.5, only emotionality remained significantly related to the outcome in the multiple analyses. Finally, deviant friends and low shyness increased the odds of EOI compared to EOD.

5. Discussion

The aim of the present study was to identify predictors of EOI among Norwegian adolescents and to examine whether these predictors differ from those of EOD. Results showed that EOI was predicted by a variety of temperamental, socio-economic, and family factors. Particularly strong and consistent findings were found in the domain of the adolescents’ own and their friends’ behaviour, where all included variables on norm-breaking behaviour were significantly related to EOI. None of these variables predicted EOD. Some of these variables lost statistical significance in the multivariate analyses. However, in both the separate and the multivariate analyses, low level of shyness and high friend deviancy differentiated between adolescents who had experienced EOI and those who just had EOD without experiencing intoxication. Thus, the results indicate that a temperament characterized by lower levels of shyness and high level of risk exposure in the friends’ network may be important factors in the aetiology of EOI.

Shyness, as conceptualized in the present study, represents the tendency to be inhibited and wary in new social situations. Interestingly, this trait, even as observed by others, seems to play a role for EOI. Shy adolescents may avoid social situations, such as parties, where friends are consuming larger quantities of alcohol, and where they themselves would be at risk of getting drunk. Shyness may thus function as a protective factor EOD compared to abstinent EOI compared to EOD

### Table 2

<table>
<thead>
<tr>
<th></th>
<th>EOI compared to abstinent</th>
<th>EOD compared to abstinent</th>
<th>EOI compared to EOD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Adolescent temperament</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shyness (m)</td>
<td>0.40***</td>
<td>(0.22–0.72)</td>
<td>0.95 (0.67–1.36)</td>
</tr>
<tr>
<td>Activity (m)</td>
<td>1.25</td>
<td>(0.82–1.99)</td>
<td>1.20 (0.89–1.61)</td>
</tr>
<tr>
<td>Emotionality (m)</td>
<td>1.16</td>
<td>(0.60–2.26)</td>
<td>1.78 (1.10–2.86)</td>
</tr>
<tr>
<td>Sociability (m)</td>
<td>2.10†</td>
<td>(1.12–3.89)</td>
<td>1.21 (0.81–1.80)</td>
</tr>
<tr>
<td><strong>Socio-economic factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family status (a)</td>
<td>1.09</td>
<td>(0.49–2.42)</td>
<td>1.56 (0.90–2.69)</td>
</tr>
<tr>
<td>Mother’s education (m)</td>
<td>1.05</td>
<td>(0.79–1.40)</td>
<td>1.17 (0.95–1.44)</td>
</tr>
<tr>
<td>Household economy (m)</td>
<td>0.56***</td>
<td>(0.37–0.86)</td>
<td>0.70† (0.51–0.96)</td>
</tr>
<tr>
<td>Mother’s employment (m)</td>
<td>0.90</td>
<td>(0.67–1.21)</td>
<td>1.67 (0.92–1.49)</td>
</tr>
<tr>
<td><strong>Family factors</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohol problems in the household (m)</td>
<td>1.64 (0.50–5.32)</td>
<td>1.79 (0.78–4.15)</td>
<td>0.91 (0.27–3.07)</td>
</tr>
<tr>
<td>Parenting—involve ment (a)</td>
<td>0.36†</td>
<td>(0.15–0.87)</td>
<td>0.65 (0.35–1.23)</td>
</tr>
<tr>
<td>Parenting—positive parenting (a)</td>
<td>0.66</td>
<td>(0.33–1.32)</td>
<td>0.94 (0.57–1.36)</td>
</tr>
<tr>
<td>Parenting—inconsistent discipline (a)</td>
<td>1.54</td>
<td>(0.87–2.72)</td>
<td>1.38 (0.91–2.09)</td>
</tr>
<tr>
<td>Parenting—other disciplinary practices (a)</td>
<td>1.08</td>
<td>(0.52–2.26)</td>
<td>1.21 (0.72–2.04)</td>
</tr>
<tr>
<td>Parenting—strictness/supervision (a)</td>
<td>0.16**</td>
<td>(0.04–0.60)</td>
<td>0.38 (0.13–1.09)</td>
</tr>
<tr>
<td><strong>Adolescents’ and friends’ behaviour</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adolescent smoking (a)</td>
<td>5.92***</td>
<td>(1.99–17.60)</td>
<td>2.67 (0.99–7.22)</td>
</tr>
<tr>
<td>Adolescent conduct problems (a)</td>
<td>1.17</td>
<td>(1.07–1.27)</td>
<td>1.07 (1.00–1.15)</td>
</tr>
<tr>
<td>Deviant friends (a)</td>
<td>2.92***</td>
<td>(1.64–5.20)</td>
<td>1.35 (0.76–2.41)</td>
</tr>
<tr>
<td><strong>Covariates</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender (girl = 0, boy = 1) (a)</td>
<td>0.49</td>
<td>(0.24–1.00)</td>
<td>0.56 (0.34–0.93)</td>
</tr>
<tr>
<td>Age (m)</td>
<td>1.55</td>
<td>(0.59–4.08)</td>
<td>1.60 (0.79–3.26)</td>
</tr>
<tr>
<td>Alcohol use before age 12.5 (a)</td>
<td>4.34**</td>
<td>(2.46–7.65)</td>
<td>3.74** (2.23–6.27)</td>
</tr>
</tbody>
</table>

OR = odds ratio; CI = confidence interval for odds ratio. EOI = early onset of intoxication (< 14.5 years); EOD = early onset of drinking (< 14.5 years); abstinent (< 14.5 years).
(m) = mother’s report; (a) = adolescent self-report.

*P < 0.05.
**P < 0.01.
***P < 0.001.
tendencies to seek stimulating experiences, willingness to take risk, and low levels of worry and rigidity in both social and non-social situations. Even when shy, adolescents will experience situations where they are offered large quantities of alcohol, but may be less inclined to drink large amounts, as they may have lower degrees of sensation and novelty seeking, concepts that have consistently been related to drug use (for a review, see Hittner & Swickert, 2006).

Deviant friends and adolescents’ own conduct problems were also related to EOI. In a culture such as the Norwegian, with very strict restriction of alcohol for the youth population, adolescents drinking large amounts of alcohol and being intoxicated can be considered a violation of societal and cultural norms. Intoxication experiences may therefore be related to a cluster of factors that are related to other behaviours deviant from culturally accepted norms. The correlation between young people’s substance use and involvement in a wide range of other problem behaviours is well documented (Elliott, Huizinga-, & Menard, 2012; Farrell, Danish, & Howard, 1992). This study adds to the literature by showing that these factors are in fact of particular importance for EOI, whereas they seem not to be related to EOD, which indicates that adolescents with parents who have greater knowledge of their whereabouts and who they are with, and stricter restrictions on staying out at night significantly decreased the risk of EOI. In contrast to former studies, none of the family factors related significantly to EOD. This also supports the notion that EOD and EOI might take place in different contexts. If EOD typically occurs in a family context, and is in fact encouraged by parents, we would not expect parenting practices reflecting different forms of control and discipline to have an effect. This implies that previously identified associations between family factors and EOD may be due to not distinguishing between the two types of drinking behaviour and that it is EOI and not EOD that can be predicted by family factors.

The present study was conducted in Norway, a country characterized by a strict alcohol regulation policy. The rates of alcohol use and drunkenness reported are consistent with national statistics and other studies on Norwegian adolescents (Bakken, 2014; Rossoew & Kuntsche, 2013). Not surprisingly, however, such rates are lower than rates in other European countries with a more liberal alcohol policy and drinking culture, such as Eastern Europe countries and Denmark (Hibell et al., 2012).

### 5.1. Strengths and limitations

The comprehensive set of potential risk factors evaluated, the comparison of different types of early drinking behaviour, and using population-based, prospective design with multiple informants make this study an important contribution to the literature on early onset. The results should however be interpreted in light of some limitations.

Information about the adolescent drinking context was not available in this study. Another concern is the statistical power of the analyses, as
only 43 adolescents reported EOI. However, in spite of the relatively low number of adolescents in this group, several significant associations between predictors and EOI were found, indicating that the effects that were obtained in the analyses were in fact of considerable size. The generalizability of these findings needs to be confirmed in other samples as our sample is based on adolescents in a Norwegian cultural context. Moreover, our sample is slightly overrepresented by adolescents whose mothers have higher levels of education. This represents a threat to the representativeness and findings are somewhat more uncertain in terms of people with low socio-economic status.

5.2 Implications and conclusion

In summary, EOI and EOD without getting drunk are related to different risk factors during childhood and adolescence. As most previous studies have not distinguished between EOD and EOI, the importance of temperament, family factors, conduct problems and friends’ networks for different types of early drinking experience has not been examined in detail. These findings need to be replicated using samples from different cultural contexts and the underlying mechanisms behind these associations need to be examined further. It is possible that some of the same underlying mechanisms that predispose adolescents to conduct problems, such as theft, vandalism, and involvement with anti-social peers could also make them prone to EOI. Some of this proneness might even have strong genetic components. There has been identified a genetic influence on EOI, and some of this genetic influence overlaps extensively with the genetic risk of alcohol use disorders (Ystrom et al., 2014). More studies are needed on the relative importance of EOD and EOI on the development of drinking patterns and alcohol-related problems in late adolescence and adulthood.

Because the nature of the relationship between EOI and later outcomes is still unclear, simply shifting focus from delaying EOD to a delay of EOI as a strategy to prevent alcohol related problems later in life has little evidence. However, preventing early drunkenness may be an important goal in itself, as immediate consequences of excessive alcohol use, such as alcohol related accidents, can be avoided. The findings in the present study indicate, in accordance with other research (i.e. Cleveland, Feinberg, & Jones, 2012; Rossov & Kuntsche, 2013; Scholes-Balog et al., 2013) that effective prevention strategies should target factors in multiple domains, including family and peers.

Funding

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Conflict of interest statement

None declared.

References


Predicting hazardous drinking in late adolescence/young adulthood from early and excessive adolescent drinking - A longitudinal cross-national study of Norwegian and Australian adolescents

Frøydis Enstad\textsuperscript{a*}, Tracy Evans-Whipp\textsuperscript{bc}, Anne Kjeldsen\textsuperscript{ad}, John W Toumbourou\textsuperscript{e}, Tilmann von Soest\textsuperscript{f}

\textsuperscript{a}Division of Mental and Physical Health, Department of Child Health and Development, Norwegian Institute of Public Health, Oslo, Norway. (froydis.enstad@fhi.no), (anne.kjeldsen@fhi.no).

\textsuperscript{b}Centre for Adolescent Health, Murdoch Children’s Research Institute, Victoria, Australia. (tracy.evanswhipp@mcri.edu.au).

\textsuperscript{c}Department of Paediatrics, The University of Melbourne, Parkville, Victoria, Australia

\textsuperscript{d}Bjørknes University College, Oslo, Norway

\textsuperscript{e}Deakin University, Geelong, Victoria, Australia. (john.toumbourou@deakin.edu.au).

\textsuperscript{f}Research Centre for Developmental Processes and Gradients in Mental Health (PROMENTA), Department of Psychology, University of Oslo, Oslo, Norway. (t.v.soest@psykologi.uio.no).

*Corresponding author: Frøydis Enstad, Division of Mental and Physical Health, Department of Child Health and Development, Norwegian Institute of Public Health, PO Box 222 Skøyen, N-0213 Oslo, Norway. Phone: +47 21 07 70 00; E-mail: froydis.enstad@fhi.no
Abstract

Background: Research has consistently shown that early onset of drinking (EOD) is associated with alcohol-related problems in adulthood. However, recent reviews have identified several limitations in the early onset literature, including the use of retrospective reports, insufficient control for potential confounders, ambiguous definitions of the concept, and an assumption that early onset is independent of cultural norms and national alcohol policies. This study addresses these limitations by examining whether EOD, independent of early onset of excessive drinking (EOE), prospectively predicts hazardous drinking in late adolescence/young adulthood in Norway and Australia, two countries with different drinking cultures. Methods: Data were drawn from two population-based longitudinal studies; the Norwegian Tracking Opportunities and Problems study (n = 329) and the Australian International Youth Development Study (n = 786). Data were collected prospectively from mid adolescence (14-16 years) to late adolescence/young adulthood (18-25 years) and a modified Poisson regression approach was used to estimate prevalence ratios. Adolescent self-reports included measures of EOD and EOE. Young adults completed the Alcohol Use Disorders Identification Test (AUDIT). The results were adjusted for adolescent factors; age, gender, impulsivity, hyperactivity, conduct problems, smoking, early sexual intercourse and friends’ substance use, and family factors; alcohol and drug use in the family, maternal education, family management and monitoring. Results: Hazardous drinking was identified in 46.8 % and 38.9 % of young adults in Norway and Australia, respectively. Both EOD and EOE in adolescence were significantly related to an increased risk of alcohol-related problems in late adolescence/young adulthood in both studies, even when adjusting for possible confounders. Conclusion: Our findings indicate that adolescent drinking behaviour is an indicator of alcohol-related problems in late adolescence/young adulthood, even when...
controlling for a variety of covariates. This finding is in contrast to previous research on older adults, where no association between adolescent drinking and later alcohol-related problems were found when controlling for covariates. The divergence in findings may suggest that the impact of EOD/EOE is limited to the late adolescent and young adult period. Preventing drinking in early adolescence may thus have some impact on the drinking patterns in late adolescence/young adulthood.

Keywords: Early onset of drinking, Early onset of excessive drinking, Prospective study, Hazardous drinking, Adolescence, Late adolescence, Young adulthood, Cross-national study
**Background**

Empirical studies have consistently shown that early onset of drinking (EOD) is related to later adverse alcohol-related outcomes [1-16]. Many of these studies suggest a causal relationship between EOD and subsequent problems [1-6, 10, 14]. However, recent reviews of the literature have questioned the assumption of a causal link [17-19]. In particular, it has been noted that: (a) research on EOD and its association with future adverse outcomes is largely based on adult samples asking participants to recall retrospectively their age of drinking onset, potentially introducing recall biases; (b) the few longitudinal studies measuring drinking onset prospectively typically suffer from lack of control for important covariates that may serve as confounders, such as externalising behaviours; (c) most studies do not provide information about whether having consumed small amounts of alcohol at an early age are related to adverse outcomes, or whether such associations are first seen when more substantial amounts are consumed; and (d) the association between early onset and adverse outcomes may vary according to the cultural context [17, 18, 20]. By using longitudinal data from two different countries, this study aims to provide a better understanding of the relationship between early drinking behaviours and hazardous drinking in late adolescence/young adulthood.

As thoroughly demonstrated in the review by Kuntsche et al. [17] the literature in this field suffers from poor reliability of the early drinking measurements as many studies are based on retrospective reports introducing potential bias. The concerns are related partly to the tendency of forward telescoping [21], as respondents tend to report the age of onset closer to their current age when interviewed. Moreover, heavy drinkers are to a greater degree biased to report an early onset of drinking than light drinkers when age of drinking is assessed retrospectively [22]. The association between retrospectively reported onset of
drinking and adult drinking behaviour may thus be explained by methodological artefacts. It has therefore been argued that prospective studies from adolescence, close in time to when onset typically occurs, are more suited to examine EOD and its relation to later alcohol-related problems [16]. Furthermore, individual level variables (e.g., conduct problems, impulsivity-hyperactivity, antisocial peer environment, health risk behaviour), and family level variables (e.g., alcohol and drug use in the family) need to be included as covariates. Developmental theories have pointed to their relevance for the development of substance use disorders [23] and empirical research link these same factors to early onset of alcohol use [3, 8, 16, 24]. Such factors are potential confounders, as they may reflect shared vulnerability to both early onset and adult alcohol-related problems.

Our review of the literature identified 15 prospective studies addressing the impact of EOD on later drinking patterns or alcohol-related problems. Among these, four followed the participants only to mid adolescence [7, 25-27] and five studies did not control for important confounding variables, such as conduct problems or parental alcohol and substance use [3, 14, 28-30]. Results from the remaining six studies following participants from early adolescence to adulthood, and with comprehensive control for possible confounders, were mixed: Whereas four studies found that the association between EOD and adult alcohol-related problems was completely explained by relevant covariates [16, 31-33], two other studies found that the association between EOD and heavy alcohol consumption remained, even after comprehensive control for potential explanatory factors, including conduct problems and parental drinking patterns [4, 11]. Thus, despite the growing number of prospective studies in this field, findings are still diverging, which may be related to varying operationalisations of early drinking by researchers, and different cultural contexts across studies.
Most of the studies in the early onset literature conceptualize onset as having had more than a few sips on one or more occasions [1, 4, 6, 14, 15, 27]. However, scholars have recently challenged the notion that consuming small amounts of alcohol in itself should have such profound impact on adolescent development [17, 34]. If the association between early drinking and adult alcohol-related problems is based on biological mechanisms, for example by early alcohol use affecting the cognitive functioning of the brain [i.e. 35] or disrupting maturational processes [36], it is more plausible to assume that such changes would occur only if the amount of alcohol consumed had a potential neurotoxic effect. Likewise, if the mechanism is of a social nature, for example through changing the identity or social role of those drinking early [i.e. 14], one could assume that such changes would be triggered only if the amount consumed violates a cultural expectation of acceptable behaviour. If the adolescents’ first encounter with alcohol is in a cultural setting where moderate alcohol use is the norm, e.g., a family dinner, it may be rather improbable that such behaviour would trigger a different developmental trajectory. Underage excessive drinking on the other hand, is a more clear breach of socially accepted behaviour, and as such has more potential to trigger a change in identity or role. A recent review supports this explanation, concluding that there are larger and more important effects observed in relation to the onset of regular drinking and experiences of intoxication than age of first drink [18].

To our knowledge, only two longitudinal studies have directly addressed whether EOD independently of early onset of excessive drinking (EOE) serves as a risk factor for later alcohol problems. First, Warner and White [33] found that the only onset related variable associated with adult alcohol problems, after a rigorous control of possible confounders, was feeling drunk at initiation. This study however only addresses different aspects of the very first drinking episode (i.e., in or outside a family context, early versus late
onset, experience pleasure or not, and feelings of drunkenness). Second, Morean et al. [12] examined age of onset and delay to first intoxication as independent predictors of heavy drinking in college students. The results showed that both an early onset and a short delay independently predicted heavy drinking, indicating that EOE confers unique risk relative to EOD, but that both factors are related to increased risk. However, the study was based on retrospective reports of drinking onset and did not control for indicators of externalizing problems or other risk taking behaviour. Thus, knowledge on whether EOE foreshadows a different drinking pattern in young adulthood from EOD, or whether any early exposure to alcohol is detrimental, is limited.

Cultural norms and national alcohol policies may also be of importance in understanding the relationship between early drinking behaviours and adult alcohol use [37]. Whilst excessive drinking and drunkenness in adolescents is likely to be viewed as a social problem in most cultures, the degree to which low levels of adolescent drinking, particularly within adult-supervised contexts, is viewed as problematic varies largely across countries and cultures. Research has shown that in permissive cultures where moderate drinking among adolescents is culturally normative, EOD is to a lesser degree linked to other markers of deviance, such as conduct problems, than in cultures were EOD is considered a greater social problem, such as the United States, where most of the research on EOD has been conducted [20]. Underage excessive drinking, on the other hand, may be more consistently related to negative outcomes across cultures. Although EOD and EOE take on meaning in the larger cultural and political context, no previous study has applied a cross-national design when examining possible associations. The present study uses data from Norway and Australia, countries with different alcohol policies and drinking cultures.
Of particular interest, prevention strategies aimed at youths differ considerably in the two countries. Norway has one of Europe’s most restrictive alcohol policies [38] with an emphasis on reducing use through reducing the demand and availability [39]. As a consequence, underage drinking is illegal with no exceptions, and the prohibition also applies to supplying minors with alcohol. Information material aimed at youth highlights activities to increase factual knowledge about alcohol, strengthening the individual’s ability to resist peer pressure and facilitating get-togethers where no alcohol is served [40]. In contrast, the overarching goal of Australia’s strategy during the study period was to minimize harm associated with alcohol use [41]. Information strategies aimed at adolescents included advice on how to stay safe and ‘in control’ when drinking [42]. The per capita consumption in Norway is among the lowest in the Western countries both among adolescents and in the adult population [43, 44], whereas Australia is among the highest among the adult population [44]. The prevalence of heavy episodic drinking is about the same in the two countries. However, somewhat paradoxically, the Norwegian drinking pattern, i.e., how they drink rather than how much, is characterized as more risky than the Australian [44]. Cultural attitudes towards underage drinking may influence associations between early drinking behaviours and later detrimental outcomes. One could hypothesize that the relationship between EOD and later problem drinking is weaker in Australia than in Norway, because EOD in Norway may represent a greater violation of formal rules and as such is more closely related to other problem behaviours which are associated with hazardous drinking in adulthood.

In summary, the present study’s primary aim is to examine the association between EOD and the association with risk of alcohol-related problems in late adolescence/young adulthood. In particular, this study addresses several limitations of most previous research by
examining (a) whether EOD (14-16 years) prospectively predicts risk of alcohol-related problems in late adolescence/young adulthood (18-25 years), (b) whether this relationship remains upon adjustment for a comprehensive number of covariates that may function as confounders, (c) whether early onset of more than a few sips of alcohol is predictive of later alcohol problems or whether only the consumption of more excessive amounts of alcohol in early age is related to such risk, and (d) whether relationships between EOD and subsequent risk of alcohol-related problems are similar in samples drawn from two different countries with different alcohol policies and drinking cultures.

Methods

Study Design and Setting
Data were obtained from the Tracking Opportunities and Problems (TOPP) study and the International Youth Development Study (IYDS); two independent longitudinal samples of adolescents and young adults in the eastern part of Norway (henceforth termed NOR) and Victoria, a southern state in Australia (henceforth termed AUS), respectively.

The participants in TOPP were recruited in 1993 when the families attended their toddlers’ 18 month vaccination at child health clinics. Originally 1,081 families from 19 geographical health care districts in eastern Norway (28 % living in large cities, 55 % in densely populated areas and 17 % in rural areas), were invited to the study, of whom 86.9 % participated in the first wave. Details of the study are described elsewhere [45]. Data used in this paper were obtained from surveys of the participants and their mothers conducted in 2006 (t6; henceforth termed adolescence, median age = 14.6, range 14.0 – 15.8) and 2011 (t8; henceforth termed late adolescence/young adulthood, median age = 18.9, range 18.3 - 19.9). The overall response rate at t6 and t8 was 49 % (n=458) and 47 % (n=441) respectively, based on participants at t1 (n=939, 87 % of the eligible sample). The final sample, with data
at both time points of interest were available for 329 young people (131 males and 198 females). Thus, from the 1,081 families invited to the study, 30.4% were included in the present study. Previous attrition analyses have shown that dropout was predicted by low maternal education level and being male [46, 47]. The study was approved by the Regional Committees for Medical and Health Research Ethics.

The IYDS is an ongoing binational longitudinal study initiated in 2002 of three cohorts (t1 in grade 5, grade 7 and grade 9) of young people in Victoria, Australia and Washington State, United States. This study uses data from the Australian sample only as data on young adults in the US was not available at the time of analysis. The oldest age (grade 9) cohort was used as this best matched the ages at which TOPP data collection was conducted. The IYDS is a school-based survey with a state wide representative sample recruited and surveyed in 2002 and followed up to 8 subsequent waves. Details of the study and school recruitment procedures has been described elsewhere [48]. In the IYDS oldest cohort, 1,288 students were eligible to participate, of whom 973 (76%) participated at t1 and 788 in the follow-up (81% retention). Honesty criteria based on responses to the t1 survey was used to remove 2 participants and so the final sample with data at both time points was 786. The surveys included in this study were conducted in 2002 (t1; henceforth termed adolescence, median age = 14.9, range 13.8 - 16.2) and 2010 (t7; henceforth termed late adolescence/young adulthood, median age = 22.9, range 21.6 - 24.6). The IYDS also includes mother report on education level from a phone survey conducted in 2002.

**Measures**

In NOR and AUS, *hazardous drinking* in late adolescence/young adulthood was assessed using the Alcohol Use Disorders Identification test (AUDIT) [49]. The AUDIT is a widely
used 10 item screening tool to identify persons with hazardous (i.e., those who are at risk of alcohol-related problems) and harmful (i.e., those experiencing some alcohol-related problems) patterns of alcohol consumption. The scores of the AUDIT range from 0 to 40, and the generally accepted cut-off for hazardous drinking is 8 and above, and a cut-off for harmful use is 16 and above [49-51]. The present study examines only hazardous use. In the NOR questionnaire, Item 3 differs from the original AUDIT in that it asks “How often do you have five or more drinks on one occasion” instead of six. Cronbach’s alpha for the whole scale was .77 (NOR) and .83 (AUS). In the NOR sample, drinking smaller amounts of alcohol and excessive use in adolescence were measured by adolescent self-report using two items; “Have you ever tasted more than a few sips of alcohol?” and “During the past 12 months, have you had so much to drink that you felt clearly intoxicated?” with five response categories (Never, Once, 2-5 times, 6-10 times and, More than 10 times). Responses were reclassified into two dummy variables; EOD relative to abstinent (have tasted one or more times, but not been intoxicated past 12 months, EOE (tasted one or more times and been intoxicated one or more times past 12 months). In the AUS sample these concepts were measured by adolescent self-report using three items; “In your lifetime, have you ever had more than just a few sips of an alcoholic beverage (like beer, wine or spirits)?” with five response categories (Never, 1 or 2 times, 3 to 5 times, 6 to 9 times, 10 or more times), “Think back over the past 2 weeks. How many times have you had five or more alcoholic drinks in a row?” (None, Once, Twice, 3-5 times, 6-9 times, 10 or more times) and “How often over the past year has your alcohol use caused you to get so drunk you were sick or passed out?” with items rated on an 8-point scale from “never” to “40 times or more”. Responses were reclassified into two dummy variables; EOD relative to abstinent (one or more times of lifetime alcohol use, but never binged and never sick or passed out), EOE (one or more times
of lifetime alcohol use and one or more times binged and/or one or more times sick or passed out).

*Hyperactivity* (NOR) was measured with a subscale of the Strengths and Difficulties questionnaire (SDQ) [52] with five items (sample items: “I am restless”, “I find it hard to sit down for long”, “I think before I do things”) with four response categories ranging from 3 (Fits very well) to 0 (Doesn’t fit at all) (Cronbach’s alpha = 0.71). *Impulsivity* (AUS) was measured by three items describing typical ways to act (i.e., thinking before acting, rushing into things, answering before thinking). Response options were NO! (1), no (2), yes (3), YES! (4) (Cronbach’s alpha = 0.57, Cronbach’s alpha based on polychoric correlation = 0.63). *Mother’s education level* (NOR) was measured by asking the mothers to indicate their highest level of education on a five point scale ranging from 1, “9 year primary school or less” to 5, “More than 4 years at college or university” and by mother report (AUS) on a three point scale of “Less than secondary school (Year 12)” (1), “Completed secondary school (Year 12)” (2) to “Completed post-secondary education (3). *Poor family management* (NOR) was assessed using adolescent-report on the short version of the “Keeping tabs” questionnaire developed for the NICHD Study of Early Child Care and Youth Development (SECCYD). The 6-items are questions regarding their parents’ supervision and monitoring (i.e. “how much does a parent know about…”; “who you spend time with?”, “…how you spend your money?”, “…where you go after school?”). Responses ranged from 1 to 4 (“knows everything” to “doesn’t know at all”) and indicate the extent to which the parent is thought to know about different aspects of the child’s whereabouts and day to day activates (NOR, Cronbach’s alpha = .85). In AUS, poor family management was measured by a nine item scale asking adolescents’ agreement to statements such as “My parents ask if I’ve gotten my homework done”, “The rules in my family are clear”, “If you skipped school without your
parents’ permission, would you be caught by your parents?”. Response options were: NO! (4), no (3), yes (2), YES! (1) (AUS, Cronbach’s alpha = 0.77). Alcohol and drug use in the family was defined to have occurred if the adolescents reported having experienced that “one of the people closest to me uses too much alcohol, pills or other drugs” one or more times during the past 12 months at t5 and/or t6 (0=No, 1=Yes) (NOR) and “Has anyone in your family ever had a severe alcohol or drug problem (0=No, 1=Yes) (AUS). Past year conduct problems was assessed by a sum score of 19 adolescent-report items taken from three Scandinavian scales of antisocial behaviour (NOR). The items covered stealing, verbally and physically aggressive behaviours, loitering, vandalism, and questions about carrying weapons. The construction of the scale is described in detail elsewhere [53]. The answers were given on an ordinal frequency scale and for this study transformed to a dichotomous (yes/no) variable, creating a variable ranging from 0 to 18 (Cronbach’s alpha = .84). In the AUS sample, conduct problems were measured with nine items covering stealing, physically aggressive behaviour, suspension from school, arrests and questions about carrying weapons, selling drugs and being drunk or high at school. The response options ranged from “never” (1) to “40+ times” (8), (Cronbach’s alpha = 0.64). Early sexual intercourse was assessed by one item asking if the adolescents had ever had sexual intercourse (yes/no) (NOR and AUS). Smoking was assessed by a single item asking if adolescents had smoked at least once in their life (yes/no) (NOR and AUS). Friends’ substance use was assessed by adolescent report on how many of their most important friends; “drinks alcohol approximately once a week”, “have tried hashish, marijuana or other illegal drugs” (NOR) and (in the past 12 months) “how many of your best friends have; “tried alcohol (like beer, wine, or liquor/spirits) when their parents didn’t know?”, “used marijuana (pot, weed, grass)”, “used other illegal drugs (like cocaine, heroin, LSD/acid, or amphetamine/speed)” (AUS). A dummy variable was
constructed contrasting those who reported at least one friend with substance use experiences with all others. *Gender* and the *age* of the child at the time of responding to the survey were assessed. Gender was dummy-coded (female coded 0 and male coded 1) (NOR and AUS).

**Statistical Analysis**

The relationships of adolescent EOD and EOE with hazardous drinking in late adolescence/young adulthood were examined by means of a modified Poisson regression approach where Poisson regression with robust error variances were estimated. This approach has been recommended instead of binary logistic regression analysis because prevalence ratios (PR) or risk ratios are obtained, thereby providing a more intuitive estimate of the association between predictors and outcomes than odds ratios from logistic regression models [54, 55]. Moreover, Poisson regressions can easily be conducted without difficulties converging. In the present study, the two dummy variables for EOD and EOE were simultaneously included as predictors of hazardous drinking (AUDIT ≥ 8) in modified Poisson regression analyses together with age and gender. Moreover, a series of Poisson regression analyses was performed to identify associations between each potential confounder with hazardous drinking, controlling for age and gender. Finally, EOD, EOE and all potential confounders were included simultaneously in Poisson regression analyses to examine the unique associations of adolescent EOD and EOE with late adolescent/young adult hazardous drinking. To examine the robustness of our results, we re-ran all analyses by using logistic models. All continuous predictor variables were standardized. Results thus indicate the change in the outcome associated with one standard deviation change in the predictor.
The overall proportions of missing data among those who participated was low. In the NOR sample, the variable with highest level of missing was hyperactivity (4.3 %), with proportions of missingness varying from 0.3 % to 1.8 % for the remaining variables. In the AUS sample, early sexual intercourse (26.4 %), and mother’s education (7.1 %) showed rather high proportions of missingness, whereas the remaining variables had between 0.3 % and 2.5 % missing data. Multiple imputation (MI) was used to handle missing data in the predictor and confounder variables, thereby providing missing data routines that are considered the state of art and to be appropriate under missing at random (MAR) conditions [56]. As recommended, 20 complete datasets were created by imputation, incorporating all variables of interest [57]. All analysis and MI were conducted separately for the Norwegian and Australian sample. To test the robustness of our results, all analyses were additionally re-run by handling missing data by listwise deletion.

Results

Table 1 provides the descriptive statistics for the study variables. As shown in the table, a total of 60.2% (NOR) and 54.8% (AUS) of the adolescents in the study were girls. At the time of the study, 13.4% of the Australian adolescents and 10.3% of the Norwegian adolescents’ reported alcohol and drug use in the family.

Table 1 about here

The frequency of drinking behaviours in adolescence and late adolescence/young adulthood for both samples is presented in Table 2. In the AUS sample, over half of the adolescents were classified as EOD, whereas 32.2 % were classified as EOE. In the NOR sample, about one quarter of the sample was classified as EOD and 13.4 % as EOE. Norwegian late adolescents/young adults reported higher rates of hazardous drinking (46.8%)
than the Australian late adolescents/young adults (38.9%), with a slight overlap of the confidence intervals of the proportions. As there was a modification in the wording of one of the ten items in the AUDIT (Item 3) in the NOR sample, we also compared AUDIT scores between the NOR and AUS sample when excluding this item. The difference between the samples did not change substantially (mean score for the AUDIT with 10 items: NOR: 7.59 versus AUS: 7.13; mean score when excluding Item 3: NOR: 6.07 versus AUS: 5.68). A further inspection of single items of the AUDIT revealed that late adolescents/young adults in Australia reported a higher frequency of drinking (AUDIT Item 1; 2-3 times a week or more) compared to the Norwegian late adolescents/young adults (AUS: 29.7 % versus NOR: 6.5 %), while a higher proportion of the Norwegian late adolescents/young adults reported drinking larger amounts of alcohol per drinking occasion (AUDIT Item 2; five drinks or more; NOR: 64.2 % versus AUS: 36.6 %).

Modified Poisson regression analyses showed a statistically significant association of both EOD and EOE with hazardous drinking while adjusting for gender and age (Table 3, Model 1). EOE was a stronger predictor than EOD of hazardous drinking in both the NOR and the AUS sample: Prevalence ratios indicated a two to three times higher risk of hazardous drinking among those with EOE, compared to those with no early drinking experiences. EOD was also significantly associated with an increasing risk of hazardous drinking, with an about two-fold increased risk of reporting hazardous drinking, compared to abstinence. Among the possible confounding variables, significant relationships between
predictors and outcome were revealed in the individual and the family domain for both samples.

When conducting Poisson regression analyses with adjustment for all confounders, EOE, although somewhat reduced, remained a significant predictor of hazardous drinking in both samples and prevalence ratios remained marginally higher than for the relationship between EOD and the outcome (see Table 3, Model 2). Likewise, the association between EOD and hazardous drinking remained statistically significant in both samples, even though prevalence ratios were somewhat reduced when including covariates. To further examine the role of EOD and EOE relative to each other, we conducted a new set of analysis where the dummy variables for early onset of drinking behaviour were recoded to set EOD as the reference group. We could thus test whether EOD increased the risk of hazardous drinking compared to EOE, when adjusting for all covariates, but no significant difference between EOD and EOE was found (NOR: PR = 1.08, 95% CI = 0.78-1.50; AUS: PR = 1.11, 95% CI = 0.92-1.35). To explore potential differences in the predictive potential of EOD versus EOE in greater detail, a new set of regression analyses was conducted, where any form for early use (including both drinking moderate amounts of alcohol and drinking excessively) was included as one dummy variable, together with all covariates. Comparable to results where EOD and EOE were included as separate variables in the regression analyses, any form for early alcohol use was significantly related to hazardous drinking in both countries (NOR: PR = 1.65, 95% CI = 1.27-2.14; AUS: PR = 1.72, 95% CI = 1.04-2.82). Moreover, to examine whether EOE predicted later hazardous drinking over and above any form of early use, EOE was included as an additional predictor in the regression equations. In line with the previous findings, results showed early use to significantly predict hazardous drinking (NOR: PR = 1.63, 95% CI = 1.25-2.13; AUS: PR = 1.69, 95% CI = 1.03-2.78), whereas EOE did not
significantly increase the risk of hazardous drinking over and above any early use (NOR: PR = 1.08, 95% CI = 0.77-1.49; AUS: PR = 1.11, 95% CI = 0.92-1.35).

Few significant relationships were identified among the possible confounding variables in the multivariate analysis (see Table 3, Model 2). In the NOR sample, only hyperactivity and male gender significantly increased the risk of hazardous drinking. In the AUS sample poor family management, smoking and male gender remained as statistically significant predictors of hazardous drinking. We also examined whether the relationship between early drinking behaviours and hazardous drinking was gender-specific, but we found no significant interactions.

Table 3 about here

Finally, all regression analyses were re-run by handling missing data by listwise deletion and results were compared to analyses when MI was used. The analyses showed no substantial differences in the results for the two ways of handling missing data. Moreover, as another robustness check, all modified Poisson models were re-ran by using logistic regressions. The results showed similar results, but with somewhat higher odds ratios compared to prevalence ratios. Such a difference was expected, as odds ratios in general yield higher values than prevalence rates, particularly when highly prevalent phenomena, such as alcohol use, are investigated [55].

Discussion

This study used prospective, cross-national data to examine the relationship of EOD and EOE with hazardous drinking. The results showed both EOD and EOE in adolescence to be related to an increased risk for alcohol-related problems in late adolescence/young adulthood, with a
somewhat stronger association for EOE than EOD. Moreover, the associations were reduced, but remained statistically significant when controlling for a comprehensive number of potential confounders, including risk factors such as conduct problems, alcohol and drug use in the family and friends’ substance use. Even though most risk factors were operationalized somewhat differently in the two samples, the same pattern of findings was identified in both NOR and AUS – two countries with different alcohol policies and drinking cultures.

Our findings support previous longitudinal studies demonstrating that EOD/EOE is related to a high risk alcohol consumption pattern [7, 11, 14, 28-30, 33, 58]. Moreover, the marked attenuation of the relationship between EOD/EOE and alcohol-related problems when including potential confounders is also in accordance with previous empirical studies [for a review, see 18]. However, somewhat surprisingly, the association between early drinking behaviours and hazardous drinking remained statistically significant, even after control for a comprehensive number of possible confounders. Our results thus indicate that adolescents with EOE/EOD are two to three times more likely to be high risk drinkers in late adolescence/young adulthood. Such findings are in contrast to previous prospective studies in which associations between early drinking behaviour and later high risk drinking patterns were completely explained by relevant covariates [16, 31, 32], but well in line with the findings of Buchmann et al. [4] and Irons et al. [11].

The divergent findings may be explained by variations in the age at which problematic drinking patterns were assessed. Typically, in studies assessing adult outcomes at ages 25 and above [16, 31, 32], associations between early drinking behaviour and later high risk drinking were not detected. In contrast, the study of Buchmann et al. [4], Irons et al. [11], and the present study, which detected associations between early drinking behaviour and later high risk drinking assessed later age drinking at 18 to 25 years. This pattern of findings may
suggest that the effect of EOD/EOE on later high risk drinking may be particularly potent in late adolescence/young adulthood.

Excessive drinking typically reaches a peak in late adolescence/young adulthood, and then gradually decreases with increasing age, accompanied by changing roles and obligations as one moves through different life phases [59-61]. A high risk drinking pattern, in the late adolescence/young adult life phase, where excessive drinking is “normative” and highly prevalent, may to a larger degree be based on social and environmental factors – including early alcohol exposure, relative to later high risk drinking. Maintenance of an excessive drinking pattern throughout adulthood is typically restricted to a smaller group of individuals characterized by more severe and long-lasting problems [61]. This drinking behaviour may be better understood in the framework of underlying genetic liability, than in light of social and environmental factors. Such a notion is supported by several genetically informed studies showing that heritability estimates increase with increasing age of the individuals and increasing severity of alcohol-related problems [62, 63]. EOD may become a non-significant predictor of adult alcohol problems when adjusting for factors thought to reflect genetic transmission or vulnerability, such as externalizing behaviours and parental substance use. In contrast, early drinking experiences in themselves may function as an early transition to a drinking culture and may thus increase the risk for hazardous drinking in social contexts, particularly in late adolescence/young adulthood where such behaviour is considered normative.

The scientific community has over the past couple of decades discussed whether more than just a few sips of alcohol per se or having consumed more substantial amounts of alcohol at an early age are related to adverse outcomes [e.g., 12, 33, 64]. The present study shows rather minor differences in how EOD versus EOE prospectively predicts later
hazardous drinking behaviour. There could be several explanations for such a finding. First, early drinking behaviours, regardless of level of alcohol consumption, may be indicators of the same proneness to risk-taking behaviours, including drinking. However, previous analyses of predictors of EOD and EOE in the NOR sample suggest that adolescents with EOD may be less inclined to take risks than adolescents with EOE, as lower levels of shyness, conduct problems and high levels of risk exposure in friendship networks were unrelated to EOD, but prospectively predicted EOE [65]. Thus, adolescents with EOE are in fact more likely to show characteristics indicating higher proneness to risk taking behaviour than EOD adolescents.

Alternatively, drawing on neurobiological and psychosocial development literature, there may be similar biological mechanisms whereby alcohol exposure leads to later alcohol-related problems [35, 36]. Based on our findings, we cannot dismiss such a biological basis, although further investigations of these relationships with a more fine-grained measure of different levels of early alcohol exposure is warranted. Furthermore, this hypothesis would be strengthened if the relationship between early onset and alcohol-related problems more consistently demonstrated a robust relationship across different studies. Our findings are more in line with an understanding of early onset, regardless of level consumed, as an introduction to a “wet” environment, enabling a progression to a hazardous drinking pattern. Interestingly, similar patterns in the relationship between early onset and risk of alcohol-related problems were identified in both the NOR and AUS samples, despite the very different proportions of adolescent drinking. The results indicate that early alcohol use seems be cross-nationally invariant and to a lesser degree is influenced by differences in cultural or social norms and national alcohol policies. Furthermore, rates of hazardous drinking were higher in the NOR sample despite the much greater adolescent abstinence. This finding
indicates that EOD/EOE cannot fully explain hazardous drinking in late adolescence/young adulthood and that there are likely to be important contextual correlates that affect this behaviour.

**Strengths and Limitations**

By using prospective studies, we minimized the potential bias caused by retrospective recall and forward telescoping that are often noted as major limitations in the literature on early onset of alcohol use [66]. The inclusion of a wide range of potential confounding factors, consideration of the independent importance of different early drinking behaviours and the use of two samples from different cultural contexts, make this study an important contribution to the literature on consequences of early drinking patterns. However, several considerations are important to note. First, the measures of early drinking behaviours give us no indication of the actual volume of alcohol consumed, although the better differentiation of early drinking experiences into EOD and EOE improves upon previous studies. Second, the NOR and AUS studies were designed as two separate studies. Consequently, the predictor variables and covariates were assessed differently in the Norwegian and the Australian samples and are not directly comparable. For example, the higher prevalence of early adolescent drinking in the AUS sample may not necessarily reflect different drinking patterns between the countries, because drinking was assessed differently in the two samples. Another limitation is that the two samples differed in the age when hazardous drinking was assessed in “late adolescence/young adulthood”. While the Norwegian sample had just reached the legal drinking age (mean age 18.9 years), the Australian participants were on average 22.9 years old. Such differences may explain different rates of hazardous drinking in the two samples, and it is possible that the Australian participants have “settled down” somewhat as
compared to the Norwegian adolescents, who may just have moved away from home and take full advantage of their new won freedom. Still, the age at which most adolescents start to drink is very similar in many Western countries. Around 50% or more of 15–16 year olds have initiated alcohol use [67]. As such, many adolescents have several years of experience with alcohol before reaching young adulthood. Despite different measures and age ranges, the patterns identified across samples were similar, indicating a robust association across countries with different alcohol policies and drinking cultures. Third, even though a comprehensive number of potential confounders were included, we did not have access to information about parents’ drinking patterns, physiological sensitivity to the effects of alcohol or genetics. The relation between early drinking behaviour and hazardous drinking could thus be caused by unmeasured common risk factors. Another concern is that information about the adolescent drinking context was not available in this study. Fourth, some measures had somewhat low internal reliability. Particularly, the three item impulsivity scale used in the Australian sample displayed low Cronbach’s alpha, with a value of .57. Pearson’s correlations, which the Cronbach’s alpha is based on, holds an assumption of normally distributed continuous variables. The items in the impulsivity scale are on an ordinal scale, with few response categories, and a slightly skewed distribution. As such, calculation of alpha based on Pearson’s correlation may lead to underestimation of the true association. In such cases, alternative strategies to estimate reliability have been suggested, such as calculating Cronbach’s alpha based on polychoric correlations, providing a more accurate estimate of the relationship between the items on the scale [68]. In our case, such an alternative estimation strategy increased internal consistency somewhat to $\alpha = .63$. Even though it is common to find low alphas in scales consisting of few items, we cannot rule out that a more reliable assessment of impulsivity could have affected the results in our study.
Fifth, AUDIT scores for the adolescent waves were not available, thereby not providing us with the possibility to examine alcohol-related problems in greater detail or to account for already existing problems when examining the relationship between early onset of alcohol use and later alcohol-related problems. Lastly, the substantial attrition rate in the NOR sample is a major limitation of the study, as only data from 30.4 % of the originally invited families were available at both time points of interest. Although attrition rates of 50 % and more are not uncommon in longitudinal studies [69], such high attrition rates may be a source of bias. Of particular concern is the fact that attrition in the NOR sample was predicted by low maternal education level and male gender, resulting in an overrepresentation of girls and adolescents whose mothers have higher levels of education. This may represent a threat to the representativeness, and our study provides limited knowledge about how early drinking behaviours are related to later alcohol outcomes in adolescents whose mothers have a low level of education. Moreover, the selective drop-out warrants caution when interpreting the results regarding gender, maternal education and family variables, even though attrition and simulation studies based on the NOR sample have shown that selective attrition leads only to biased estimates of means of variables; estimates of associations between variables were not affected even with selective attrition and high attrition rates [70]. Still, research on these associations in low socio-economic strata will be of importance in future studies. The AUS sample is a Victoria sample, and so may be limited in its generalisability to the whole country.

Conclusion

In summary, this study used prospective, cross-national data to examine the relationship of EOD and EOE with hazardous drinking in late adolescence/young adulthood. Our findings
indicate that early drinking behaviour, regardless of level, is an indicator of alcohol-related problems in late adolescence/young adulthood even when controlling for a variety of covariates. This pattern is consistent across samples drawn from two countries with different prevention policies and drinking cultures. Our findings are in contrast to previous research on older adults, where no association between adolescent drinking and later alcohol problems was found when controlling for covariates. The divergence in findings may suggest that the impact of EOD/EOE is limited to the late adolescent and young adult period. We suggest that EOD and EOE play a role first and foremost as an introduction to a drinking culture enabling a progression to a hazardous drinking pattern. Efforts to reduce early age drinking and reduce the progression from experimental/minor alcohol use to instances of EOE may have some impact on the risk of alcohol-related problems in late adolescence/young adulthood. Future research should examine the role of early drinking behaviours and indicators of alcohol-related problems in different life phases.

**Abbreviations**

EOD: Early onset of drinking; EOE: Early onset of excessive drinking; AUDIT: Alcohol Use Identification Test; NOR: Norway/Norwegian; AUS: Australia/Australian; PR: Prevalence ratios; CI: Confidence interval.

**Declarations**

**Ethics approval and consent to participate**

The Norwegian TOPP study has been approved by the Norwegian Data Protection Authority and the Regional Committees for Medical and Health Research Ethics (REC) South East Norway. The TOPP study started in 1992/1993 when written consent in research studies was
not a requirement. However, in later years, REC has reviewed all information given to participants and concluded that the study fulfils essential requirements for informed consent. Study participation was voluntary. Information about the study, the possibility to skip questions, and the right to withdraw from the study at any point, was provided in writing to both the children and their mothers for each data collection. The children were invited through their mothers, who were given questionnaires and separate envelopes for themselves and their children. In the Australian IYDS study, ethics approval was first gained through the Ethics in Human Research Office at the Royal Children’s Hospital in Victoria. At time 1, permission was then gained from the Victorian Department of Education and Training for government schools and the Catholic Education Office for some private schools and then by school principals. Parents provided written consent for their child to participate in the study and students provided assent to complete the survey. At time 7, ethics approval was gained through The University of Melbourne Human Ethics in Research Committee and young adult participants provided online assent at the time of survey completion.

**Consent for publication**

Not applicable.

**Availability of data and material**

The TOPP study provides access to data for all members of the TOPP study. Data for external researchers can only be made available on request, provided that the TOPP-research group have available resources to facilitate and administer collaboration. The IYDS study provides limited access to approved collaborators. Core researchers of the IYDS have
permission to use the data, but data for external researchers can only be made available on request provided that the IYDS principal investigators have approved the collaboration.

**Competing interests**

The authors declare that they have no competing interests.

**Funding**

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**Authors' contributions**

FE and AK assisted in the management of several TOPP data collections. TEW and JT assisted the management of IYDS data collection. FE and TEW prepared the data, and FE performed the statistical analyses. Authors FE, TVS and TEW designed the study, FE led the drafting of the manuscript, and all other authors contributed interpreting the results and drafting the manuscript. All authors critically reviewed the manuscript and approved the final version.
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Table 1. Descriptives of study variables

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Standard deviation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Norway (n = 329)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Girls (a), %</td>
<td>60.2</td>
<td></td>
</tr>
<tr>
<td>Age (m)</td>
<td>14.58</td>
<td>0.31</td>
</tr>
<tr>
<td>Hyperactivity (a)</td>
<td>1.13</td>
<td>0.53</td>
</tr>
<tr>
<td>Conduct problems (a)</td>
<td>2.60</td>
<td>2.87</td>
</tr>
<tr>
<td>Early sexual intercourse (a), %</td>
<td>8.3</td>
<td></td>
</tr>
<tr>
<td>Smoking (a), %</td>
<td>25.4</td>
<td></td>
</tr>
<tr>
<td>Friends’ substance use (a), %</td>
<td>43.3</td>
<td></td>
</tr>
<tr>
<td>Mother’s education (m)</td>
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<tr>
<td>Primary school (9 years or less), %</td>
<td>3.4</td>
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<tr>
<td>Secondary school (1-3 years), %</td>
<td>37.5</td>
<td></td>
</tr>
<tr>
<td>Higher education (≤4 years), %</td>
<td>25.7</td>
<td></td>
</tr>
<tr>
<td>Higher education (&gt;4 years), %</td>
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<td>Poor family management (a)</td>
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<td>0.58</td>
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<tr>
<td>Alcohol and drug use in the family (a)</td>
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<td></td>
</tr>
<tr>
<td><strong>Australia (n = 786)</strong></td>
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<td></td>
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<tr>
<td>Girls (a), %</td>
<td>54.8</td>
<td></td>
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<tr>
<td>Age (m)</td>
<td>14.89</td>
<td>0.38</td>
</tr>
<tr>
<td>Impulsivity (a)</td>
<td>2.06</td>
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<tr>
<td>Conduct problems (a)</td>
<td>1.12</td>
<td>0.30</td>
</tr>
<tr>
<td>Early sexual intercourse (a), %</td>
<td>11.1</td>
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<tr>
<td>Smoking (a), %</td>
<td>53.0</td>
<td></td>
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<tr>
<td>Friends’ substance use (a), %</td>
<td>80.0</td>
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<tr>
<td>Mother’s education (m)</td>
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<tr>
<td>Primary school, %</td>
<td>42.2</td>
<td></td>
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<tr>
<td>Completed secondary school, %</td>
<td>31.9</td>
<td></td>
</tr>
<tr>
<td>Higher education, %</td>
<td>26.1</td>
<td></td>
</tr>
<tr>
<td>Poor family management (a)</td>
<td>1.86</td>
<td>0.48</td>
</tr>
<tr>
<td>Alcohol and drug use in the family (a), %</td>
<td>13.4</td>
<td></td>
</tr>
</tbody>
</table>

Note. Variables are measured differently in the Norwegian and Australian samples and values are therefore not directly comparable. (a) = Adolescent self-report; (m) = Mother’s report.
Table 2. Proportion of early onset drinking (EOD), early onset excessive drinking (EOE), abstinent (14-16 years) and hazardous and non-hazardous drinking in late adolescence/young adulthood (18-25) in the Norwegian and Australian sample.

<table>
<thead>
<tr>
<th>Alcohol use in adolescence</th>
<th></th>
<th>Norway</th>
<th></th>
<th>Australia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>95% CI</td>
<td>n</td>
</tr>
<tr>
<td>EOD</td>
<td>84</td>
<td>25.5</td>
<td>20.8-30.2</td>
<td>442</td>
</tr>
<tr>
<td>EOE</td>
<td>44</td>
<td>13.4</td>
<td>9.7-17.1</td>
<td>253</td>
</tr>
<tr>
<td>Abstinent</td>
<td>201</td>
<td>61.1</td>
<td>55.8-66.4</td>
<td>91</td>
</tr>
<tr>
<td>Alcohol use in late adolescence/young adulthood</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-hazardous drinking (AUDIT &lt; 8)</td>
<td></td>
<td>173</td>
<td>53.2</td>
<td>477</td>
</tr>
<tr>
<td>Hazardous drinking (AUDIT ≥ 8)</td>
<td></td>
<td>152</td>
<td>46.8</td>
<td>304</td>
</tr>
</tbody>
</table>

Note. EOD, EOE and abstinence are measured differently in the Norwegian and Australian samples and prevalences are therefore not directly comparable.

95% CI = 95% confidence interval of percentage estimate.
AUDIT = Alcohol Use Disorders Identification Test.
Table 3. Results of modified Poisson regressions predicting hazardous drinking (AUDIT ≥ 8).

<table>
<thead>
<tr>
<th>Individual variables</th>
<th>Hazardous drinking (AUDIT ≥ 8) Norway</th>
<th>Hazardous drinking (AUDIT ≥ 8) Australia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Model 1(^{a})</td>
<td>Model 2(^{b})</td>
</tr>
<tr>
<td>PR (95% CI)</td>
<td>PR (95% CI)</td>
<td>PR (95% CI)</td>
</tr>
<tr>
<td>----------------------</td>
<td>----------------------</td>
<td>----------------------</td>
</tr>
<tr>
<td><strong>EOD relative to abstinent (a)</strong></td>
<td>1.78 (1.39-2.30)*****</td>
<td>1.63 (1.25-2.13)*****</td>
</tr>
<tr>
<td><strong>EOE relative to abstinent (a)</strong></td>
<td>2.10 (1.52-2.66)*****</td>
<td>1.76 (1.19-2.60)****</td>
</tr>
<tr>
<td>Impulsivity (a) (z)</td>
<td>-----</td>
<td>-----</td>
</tr>
<tr>
<td><strong>Hyperactivity (a) (z)</strong></td>
<td>1.26 (1.13-1.42)*****</td>
<td>1.17 (1.03-1.33)*</td>
</tr>
<tr>
<td>Conduct problems (a) (z)</td>
<td>1.24 (1.14-1.34)*****</td>
<td>1.11 (0.97-1.26)</td>
</tr>
<tr>
<td>Early sexual intercourse (a)</td>
<td>1.16 (0.79-1.71)</td>
<td>0.72 (0.48-1.08)</td>
</tr>
<tr>
<td>Smoking (a)</td>
<td>1.28 (1.01-1.63)*</td>
<td>0.92 (0.71-2.18)</td>
</tr>
<tr>
<td>Friends' substance use (a)</td>
<td>1.31 (1.04-1.65)*</td>
<td>0.90 (0.69-1.19)</td>
</tr>
<tr>
<td>Gender (girl = 0, boy =1) (a)</td>
<td>1.37 (1.09-1.72)****</td>
<td>1.36 (1.07-1.71)*</td>
</tr>
<tr>
<td>Age (m) (z)</td>
<td>1.00 (0.89-1.13)</td>
<td>0.98 (0.88-1.00)</td>
</tr>
<tr>
<td><strong>Family variables</strong></td>
<td></td>
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<tr>
<td>Mother's education (m) (z)</td>
<td>1.03 (0.91-1.16)</td>
<td>1.03 (0.92-1.16)</td>
</tr>
<tr>
<td>Poor family management (a) (z)</td>
<td>1.20 (1.07-1.34)****</td>
<td>1.02 (0.90-1.17)</td>
</tr>
<tr>
<td>Alcohol and drug use in the family (a)</td>
<td>1.51 (1.14-2.01)****</td>
<td>1.13 (0.82-1.53)</td>
</tr>
</tbody>
</table>

Note. Variables are measured differently in the Norwegian and Australian samples and values are therefore not directly comparable. *P < 0.05; **P < 0.01; ***P < 0.001. PR = Prevalence Ratio; 95% CI = 95% confidence interval of PR.

\(a\) = adolescent self-report; \(m\) = mother’s report; \(z\) = standardized variables.

\(^a\) Separate analyses are conducted for each predictor, with control for age and gender. PR of age and gender are not controlled for covariates.

\(^b\) All predictor variables are included simultaneously.