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A cognitive model for the intergenerational transference of alcohol use behavior

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ABSTRACT

A family history of alcoholism has shown to be one of the greatest consistent risk factors in the intergenerational transference of alcohol problems. Whereas a large number of studies have attempted to identify the processes responsible for this interfamilial transfer, the mechanisms remain unclear. Family, twin and adoption studies, and environmental theories have resulted in a number of unanswered questions regarding the extent that these factors influence the transmission of alcohol behavior. Recently, cognitive theories have suggested that the observation of parental drinking habits contributes to the child's beliefs and expectations of alcohol's effects. A hypothesised cognitive model will be proposed suggesting that the mechanism for the transference of particular drinking styles from parent to offspring may be further explained by the transference of alcohol cognitions, in particular, alcohol expectancies and drinking refusal self-efficacy. This review focuses on research of bio/psycho/social factors that perpetuate alcohol misuse across generations, and will delineate the proposed cognitive mechanisms for the interfamilial transference of alcohol problems and discuss the implications of the proposed model.

Contents

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1. Introduction

Reports by the Australian Institute of Health and Welfare (AIHW, 2001) reveal that alcohol is the second greatest cause of drug-related deaths and hospitalisations in Australia, with an estimated 31,133 individuals (75% male and 25% female) dying from risky and high-risk alcohol use in the decade between 1992 and 2001. Similar results are reported by the National Drug Research Institute (Chikritzhs et al., 2003), which indicate that 85% of total alcohol consumption by females aged 14 to 24, and 80% by males aged 14 to 17 was drunk at a risky to high-risk level for acute harm (e.g. assault, road injury, drowning). The estimated economic cost to the Australian community due to alcohol abuse in one year (1998–1999) totalled $5.5 billion, with total intangible costs of just over $2 billion, which included the cost of pain and suffering ($218 million) and loss of life ($1.8 billion). As demonstrated by these statistics, alcohol misuse is costly from individual and societal levels, and understanding the mechanisms by which these behaviors are transferred is an important component in creating treatment and prevention strategies for alcohol abuse.

1.1. Intergenerational transference of alcohol symptomatology

It is clear from the children of alcoholics (COAs) literature that the intergenerational transference of alcohol problems has been well established (see the University of Missouri study e.g., Sher, 1991, 1997; Sher, Walitzer, Wood, & Brent, 1991; the University of Michigan Longitudinal Study e.g., Zucker, 1989; Zucker & Fitzgerald, 1991; and the Minnesota Twin Family Study (McGue & Iacono, 2004)). Estimated rates of alcoholism reveal that male and female alcoholics are more likely to have a father and/or mother who was alcoholic, compared to offspring in the general population (Sher, 1997), thus suggesting that parents play a major role in the acquisition of drinking problems. Although evidence shows quite convincingly that the *symptoms* of alcohol dependence aggregate within alcoholic families, it remains unclear by what *mechanisms* these symptoms are transferred, and more specifically, whether the transmission of alcohol misuse is a direct causal relationship between parental and offspring alcoholism, or due to other mediating variables such as genetic, environmental, or cognitive factors.

A number of reviews and studies have focused on the intergenerational "transfer of risk", whereby specific parental characteristics and behavior (e.g., gambling, antisocial behavior, substance abuse, domestic violence) are associated with an increase in the possibility that similar or related problems will occur in the next generation, and have aimed to determine the processes by which this transfer occurs (e.g., Blaze, Iacono, & McGue, 2008; Hicks, Krueger, Iacono, McGue, & Patrick, 2004; Oei & Raylu, 2004; Serbin & Karp, 2004; Van Ijzendoorn, 1997; Velleman, 1992). In alcohol literature, various theories have been proposed for the intergenerational transference of alcohol problems, however the existing dominant explanation for this phenomenon is genetic theory, which proposes that problematic drinking behaviors are transferred from parent to child through biological processes. Nonetheless, this theory has resulted in a number of unanswered questions, suggesting that other influences may also play a part in the transference of the behavior. For example, environmental theories suggest that influences like family dysfunction and social class explain a large proportion of the risk for heavier drinking and alcohol-related problems (Velleman, 1992). Yet, as with genetic theories, the extent that these factors influence the transmission of alcohol behavior within families remains debatable, leaving a large proportion of variance in interfamilial alcohol transference unaccounted for.

More recently, cognitive theories of transference have become more prominent and argue that information regarding alcohol and its subsequent effects are acquired during childhood, and that parents have a major impact on these acquisition processes and outcomes. In particular, cognitive theories propose that the observation of parental drinking habits contributes to the child's beliefs and expectations of alcohol's effects, which in turn reinforces their future alcohol use behavior. It is suggested that the acquired alcohol information is retained in the child's long-term memory, and then triggered once alcohol use begins.

To date, the literature outlining the mechanisms for the transmission of alcohol problems within families has not been systematically reviewed and as such the current paper will review these theories and, based on results from previous research, will propose that cognitions (rather than direct observation and imitation) between parents and offspring act as a mechanism to explain the intergenerational transference of alcohol problems.

1.2. The current review — issues addressed

The three primary aims of the current paper are; 1) to review the existing research for the intergenerational transference of alcohol problems, 2) to delineate the proposed mechanisms for this transmission within high-risk families by evaluating the nature and extent of genetic, environmental, and psychosocial risk, as outlined in the literature, and 3) to present a hypothetical structural model incorporating the behavioral and cognitive influence of the parents in the intergenerational transference of alcohol use.

Potential causes or influences of alcohol abuse/dependence[1] will be reviewed, focusing in particular on bio/psycho/social factors leading to the perpetuation of alcohol misuse across generations. The review will begin by examining familial resemblance of alcohol abuse and dependence symptoms using evidence from family, twin and adoption studies, before a discussion of the potential moderation of parent and child gender differences. Subsequently, the paper will examine various proposed mechanisms to explain the interfamilial transference of alcohol problems beginning with a brief review of biological studies that have examined similarities in the human genome within families. This will be followed by an evaluation of the significance of genetic and shared environmental influences, and how these factors may interact to increase the risk of alcohol problems within high-risk families. Consequently, a brief assessment of the research into the potential direct and indirect risk factors for alcohol misuse encountered within dysfunctional family units will be covered. This section will aim to determine if such adversity (e.g., domestic violence, poor parenting) adds to the variability in alcohol abuse/dependence in these families, above and beyond their co-existence with alcohol problems in parents. Finally, the review will conclude with a more comprehensive assessment of cognitive studies, with a particular focus on research that has assessed the interfamilial transmission of alcohol expectancies. This section will provide the background for the second aim of the paper, which is to introduce the proposed cognitive model suggesting that the mechanism for the transmission of particular drinking styles from parent to offspring may be further explained by the transference of alcohol cognitions, in particular, alcohol expectancies and drinking refusal self-efficacy.

A search of PsychINFO and MEDLINE was conducted between the years 1980 and 2007, using the following keywords as identifiers: *intergenerational*, *transference*, *transmission*, *parent*, *child*, *offspring*, *family*, *twin*, *adoption*, and *alcohol*. The reference sections of articles acquired by the electronic search were also used to identify additional research reports that contained information on interfamilial alcohol use disorders. Articles assessing the effect of alcohol use on the foetus during pregnancy, offspring alcohol problems resulting from other

[1] These terms, together with 'alcohol problems', 'alcohol misuse', and 'problem drinking' are often subsumed (either correctly or incorrectly) under the umbrella term 'alcoholism'. These terms will however be used interchangeably throughout this paper, unless referring directly to the original studies cited, where the original authors' terms will be used.

parental psychopathy, parental alcohol misuse leading to other offspring problems (e.g., illicit drug use, mental disorders), and papers assessing the transference of alcohol problems within cultural groups' customs, values, or religious influences were excluded from the current review.

2. Familial similarities for alcohol use disorders

The following sections will demonstrate the familial resemblance of alcohol use in family, twin and adoption studies. The purpose of this discussion is to outline those studies that have revealed interfamilial similarities for alcohol abuse/dependence symptomatology between first-degree relatives, and how this provides evidence for the intergenerational transference of alcohol problems.

2.1. Family studies

The familial aggregation of alcohol problems has been demonstrated through various family, twin, and adoption studies and results from alcoholism research have shown that a family history of alcohol abuse/dependence is one of the best explanatory predictors of the initiation and maintenance of later problem drinking behavior, in both clinical (Hartman, Lessem, Hopfer, Crowley, & Stallings, 2006; Hill & Yuan, 1999), and community samples (Chassin & Barrera, 1993; Lieb et al., 2002; Sher, 1991; Webster, Harburg, Gleiberman, Schork, & DiFranceisco, 1989). The Yale Family Study of clinic-referred families has revealed that a family history of alcohol abuse/dependence contributed a significant proportion (43%) of the variance in their relative's problematic alcohol symptoms, which was further increased (60%) with the familial concentration of alcohol problems (Conway, Swendsen, & Merikangas, 2003). That is, the more relatives with a history of alcohol problems, the greater the risk of their relatives displaying problematic drinking behaviors. Similarly, in a study of 8296 first-degree relatives of alcoholic probands, alcohol dependence was found to be two to three times greater than that reported by controls, based on four diagnostic criteria (i.e., DSM-III-R, DSM-IV, ICD-10, and Feighner et al., definite alcoholism criteria), with overall lifetime risk rates for alcohol dependence of 28.8% for relatives of probands, and 14.4% for controls (Nurnberger et al., 2004). Another treatment sample used male adolescents diagnosed with alcohol abuse or dependence, and results revealed that 33% of the variance in alcohol abuse and 56% in alcohol dependence were attributed to factors transmitted from parents (Hartman et al., 2006). Hartman et al., argue however that the use of clinical samples may include more severe cases of alcohol abuse/dependence and higher levels of comorbidity with other psychiatric diagnoses, and may therefore not generalise to alcohol problems in the general population (Hartman et al., 2006).

In a community sample, Lieb et al. (2002) found that the presence of AUDs in both parents elevated the risk of their children consuming alcohol in higher use categories compared to those who had only one or no affected parents. These results were paralleled in a more recent study by Chalder, Elgar, and Bennet (2006) who found that offspring of parents with alcohol problems reported drinking more frequently and at higher amounts than their counterparts from families with no reported alcohol-related problems. These children were also more likely to report coping, enhancement, and conformity among their motivations to drink, and were also likelier to drink alone, drink to induce intoxication, and drink due to the pleasurable taste of alcohol.

Chalder et al.'s findings may however be explained by those of Sher et al. (1991) who revealed that although children of alcoholics reported heavier alcohol consumption, greater dependency symptoms, and were more likely to receive a lifetime alcohol diagnosis than those children without alcoholic parents, when evaluating the relationship between family history of alcoholism and alcohol involvement with other variables thought to be etiologically relevant to the development of alcohol problems (e.g. alcohol expectancies, personality, drug use, psychopathology) it was found that the relationship between family history and alcohol involvement was mediated by behavioral undercontrol (e.g. hyperactivity, impulsivity, extraversion, antisociality, sensation seeking) and alcohol expectancies.

Various studies have revealed that there is not only an association between parental alcohol misuse and offspring alcohol problems, but also between abstentious and low/medium alcohol use of parents (Harburg, DiFranceisco, Webster, Gleiberman, & Schork, 1990; Webster et al., 1989). These findings have shown that the relationship between parent and offspring alcohol consumption varies according to parental drinking patterns. Nonetheless, it is important to recognise that alcohol misuse was noticeably elevated in offspring if a parent drank heavily and even more so if both parents drank in excess.

This research provides strong evidence for the transference of alcohol problems from parents to offspring, and provides support to the theory that underlying mechanisms function within families to expedite this transference. However, it has been argued that the use of "at risk" family studies fails to recognise the influence of genetic and environmental factors in the etiology of relative's drinking compared with twin or adoptee studies where more observable genetic markers are available (Walters, 2002).

2.2. Twin studies

Twin studies, like family studies, have also shown that a family history of alcoholism is a consistent risk factor in developing alcohol dependence (Han, McGue, & Iacono, 1999; Heath, 1995; Merikangas et al., 1998). These studies compare similarities between monozygotic (MZ) and dizygotic (DZ) twins of alcoholic parents raised together, and allows for investigations of genetic and environmental influences between individuals with 100% shared genes and those who share only 50% of their genes. Various studies of alcohol abuse/dependence have demonstrated the familial nature of such disorders with MZ–DZ concordance ratios of approximately 2:1 (Cleveland & Wiebe, 2003; Koopmans, Slutske, van Baal, & Boomsma, 1999; Maes et al., 1999; Prescott & Kendler, 1999; Rose, Dick, Viken, & Karpio, 2001). A major Australian twin study of over 4000 male and female MZ and DZ twin pairs has consistently shown that heritability scores for the likelihood of alcohol dependence symptoms range from 47% to 64% (Heath et al., 1997; Knopik et al., 2004; Whitfield et al., 2004). Similarly, in a study of 1514 male–male twin pairs, Prescott and Kendler (1999) revealed that 43%–58% of variance in the development of AUDs was attributed to additive genetic influences, with pair correlations for alcohol abuse or dependence (DSM-IV criteria) of 55% and 31% for MZ and DZ twins, respectively. These similarities were also reflected in an analysis of female–female twin pairs, which revealed the heritability of alcoholism risk in the range of 50% to 60% (Kendler, Heath, Neale, Kessler, & Eaves, 1992). Furthermore, whereas Heath et al. (1997) have found that AUD concordance rates were substantially higher for male than for female twins, their results also revealed a significantly elevated risk for AUDs in MZ compared to same-sex DZ pairs, both in men (56% *vs.* 33%) and women (30% *vs.* 17%), providing interfamilial similarities for alcohol abuse/dependence symptomatology in both genders.

2.3. Adoption studies

Gene–environment studies on adopted away children have frequently shown an increased risk for alcohol misuse in adoptees from an alcoholic biological background (compared to control adoptees), and revealed that alcohol abuse by the adoptive parents is not associated with greater risk of alcohol abuse in the adopted child, suggesting a minor influence of environment (Bohman, Cloninger, Sigvardsson, & von Knorring, 1987; Cadoret, Cain, &

Grove, 1980; Goodwin, Schulsinger, Hermansen, Guze, & Winokur, 1973; Goodwin et al., 1974; McGue & Sharma, 1995; Sigvardsson, Bohman, & Cloninger, 1996). Although, in an extension of these adoption studies, Newlin, Miles, van den Bree, Gupman, and Pickens (2000) also included stepfamilies with a non-alcoholic biological parent, and either an alcoholic or non-alcoholic stepparent, which enabled further scrutiny of specific hypotheses concerning genetic and environmental influences. Specifically, the inclusion of one non-alcoholic biological rearing parent with one non-biological (either alcoholic or non-alcoholic) parent can in turn exclude the genetic transmission of alcohol problems in these offspring. Their results revealed that having an alcoholic biological parent significantly increased the risk of their offspring developing alcohol abuse and dependence symptoms, with associations almost three-fold greater for biological than adoptive and step families. However, the results for non-biological families were less straightforward such that having an alcoholic adoptive mother or an alcoholic stepfather predicted offspring alcohol abuse, suggesting specific environmental transmissions. As suggested by Newlin et al., these heightened risks may possibly be attributed to other psychopathology in the biological parent, which may have contributed to the adopting out of the child, the termination of prior marriages, and/or some form of abuse or neglect by a stepparent, however, the importance of these factors as mediators of the relationship between parental and offspring alcohol misuse will be discussed in a further section.

The findings from twin and adoption studies indicate the importance of a genetic factor in the intergenerational transference of alcohol problems, yet these studies, like family studies, imply that other mediating processes are functioning to initiate the transmission of alcohol problems. Research assessing parent and child gender differences seem to support this suggestion, and imply that other mechanisms are at work rather than just direct genetic similarities or simple observation and imitation of parental role models.

2.4. Parent × child gender differences

Due to the lower prevalence of alcohol problems in females (Nolen-Hoeksema, 2004), many intergenerational studies on alcohol use have excluded mothers and daughters, and have focused predominantly on data from fathers and sons. Studies and meta-analyses that have included female data have revealed that the association between maternal and offspring alcohol problems is similar to that between paternal and offspring patterns, suggesting an equivalent genetic load for alcohol dependence in both genders (Cloninger, Bohman, & Sigvardsson, 1981; Walters, 2002).

This familial relationship however appears to be moderated by parental and offspring gender (Moser & Jacob, 1997; Pollock, Schneider, Gabrielli, & Goodwin, 1987; Van Gundy, 2002; Weinberg, Dielman, Mandell, & Shope, 1994). A number of earlier studies found that a same-sex pattern emerged whereby paternal alcohol problems were associated with greater alcohol use in their sons, whilst maternal alcohol misuse predicted daughters increased alcohol use (Harburg, Davis, & Caplan, 1982; Yu & Perrine, 1997), suggesting a modelling or imitation effect of the same-sex parent. However, more recent research has revealed that drinking in either parent increases the risk of alcohol misuse and heavy alcohol use in their children. For example, Lieb et al. (2002) revealed that maternal AUDs were associated with their offspring's progression from occasional alcohol use into regular use, whereas paternal AUDs increased the chance of their children's progression from regular to hazardous levels of alcohol use. More specifically however, Webster et al. (1989) revealed that the sons of abstemious fathers were themselves abstemious or drank at low volumes. Fathers who drank medium levels of alcohol also tended to have sons in the same alcohol use category, however, sons of fathers in the high volume range were just as likely to drink alcohol in high levels as those sons with low and medium use fathers. A different pattern of results emerged however for mothers and sons. Whereas abstemious mothers also tended to have abstemious sons, high alcohol use mothers were likelier to have sons who abstained or drank in low quantities. In contrast, low alcohol use mothers tended to have sons who drank in high quantities. The associations for fathers and daughters and mothers and daughters were similar such that daughters drank in low quantities irrespective of their parent's drinking volume category, with the exception of high use mothers who also tended to have daughters who drank alcohol in high volumes. It is worth noting however that the sample size in this category (i.e., mothers/daughters) was very small, which may have contributed to the strong association in terms of relative risk (Webster et al., 1989). Nonetheless, these results in particular imply that whereas alcohol abuse/dependence symptoms aggregate within families, other processes are operating to mediate this intergenerational transference, such that the direct effects of parental gender and alcohol use behavior on offspring alcohol use behavior were not so straightforward.

Given the often differential relationships between parent and child drinking habits and gender, it has been argued that two types of alcohol misuse patterns exist; a genetically influenced type, and another less severe type predisposed to environmental impacts (also referred to as Type II or male-limited, and Type I or milieu-limited, respectively, Cloninger, 1987). For instance, Davies and Lindsay (2004) have proposed that females are more sensitive to disruptions in their home environment, which may leave them vulnerable to alcohol misuse, whereas males are more prone to genetic influences. In support of this theory, Han et al. (1999) found that additive genetic factors accounted for approximately 60% of the variance in alcohol use in 17–18 year old Dutch twin males, but only 10% in female twins, however, shared environmental factors accounted for 68% in females alcohol use, but only 23% in males alcohol use. Ironically however, despite the large variation in these heritability estimates, the differences were not statistically significant. Additionally, the male-limited and milieu-limited hypothesis was only partially supported in a longitudinal study (Coffelt et al., 2006), which found that daughters, but not sons, demonstrated elevated rates of alcohol use in response to paternal, but not maternal, drinking behavior. Furthermore, Coffelt et al., also found no interaction between paternal and maternal alcohol misuse and child gender, indicating that cumulative risk for either offspring gender was not associated with increased risk of interfamilial transference of alcohol problems.

2.5. Summary and critique

Family, twin, and adoption studies have provided strong evidence that the symptoms of alcohol abuse/dependence aggregates in families, and the intergenerational transference of alcohol problems can be attributed, at least in part, to a genetic mechanism. Many of these analyses have reported heritability statistics of 40%–60%, with familial concentration of alcohol problems (i.e., additivity of alcohol abusing/dependent relatives) contributing to elevated levels of symptom transference. However, various problems arise insofar that "at-risk" family studies often confound biology and environment such that most families share these elements. The development of twin studies reduced this limitation, arguing that greater similarities would occur between identical twins that share 100% of their genetic material, compared to fraternal twins, who only share 50%. The heritability rates for these studies provide further support for a genetic influence in the transference of alcohol abuse/dependence symptoms, with a twofold risk of alcohol problems in identical twins, compared to non-identical twins. Similarly, adoption and stepfamily studies have revealed that alcohol problems in the biological parent, but not the non-biological parents, are frequently associated with a greater risk of alcohol problems in the adopted or stepchild. Furthermore, the examination of the moderating roles of parent and child gender has

revealed that the genetic load for alcohol dependence is equivalent in both genders, despite a vast majority of the literature excluding female data due to the lower prevalence of alcohol problems in females.

However, twin and adoption studies are not without their limitations (although the evidence is mixed), particularly in regards to assortative mating and equal environment assumptions for twin pairs, and pre/postnatal influences or selective placement for adoptees (Keller & Coventry, 2005; Walters, 2002). In a meta-analysis of 50 family, twin, and adoption studies, Walters' (2002) revealed heritability estimates much lower than those rates normally cited in alcohol literature. In fact, after restricting those studies that contribute highly to the genetic hypothesis (i.e., males diagnosed with severe alcohol dependence), the heritability scores for the interfamilial transference did not exceed 26%. Even the inclusion of the studies of alcohol dependent males only increased the estimate to 30–36%. These findings indicate that up to 70% of the variance in alcohol transference is attributable to other factors. Numerous studies have explored various biological, environmental and cognitive factors to explain the processes by which the transference of alcohol problems is attributable.

3. Mechanisms in the intergenerational transference of alcohol problems

The previous sections discussed the familial similarities in problem drinking behaviors, however the following sections will evaluate the research to date that has examined various mechanisms to explain this transfer of alcohol use/abuse behaviors. It should be noted that the current review is by no means exhaustive and many reviews have suggested that there are multiple pathways to alcohol abuse (e.g., Searles, 1988; Sher, Grekin, & Williams, 2005). Nonetheless, as family, twin, and adoption studies have consistently implicated genetic factors in the development of alcohol problems, the review will begin with research that has sought to confirm the existence of specific genetic markers to determine disease susceptibility.

3.1. Human genome studies

Family studies have indicated that the human genome influences the risk of developing alcohol problems, which has led to a plethora of research examining the effect of specific chromosomal regions and genes to further explain the mechanism for the intergenerational transference of alcohol use disorders (see Dick & Foroud, 2003; Higuchi, Matsushita, & Kashima, 2006; and Tyndale, 2003, for reviews). These include both linkage, and association studies. Linkage studies involve families with multiple affected individuals, and identify variations within segments of DNA and chromosomal regions that are common among family members (Dick & Foroud, 2003; Tyndale, 2003). The linkage concept suggests that genes located in close proximity to each other are likelier to be inherited together from one parent, than two distal genes (Higuchi et al., 2006). In contrast, association studies are not restricted to family samples and can use unrelated controls to assess the relationship between specific genes and a particular outcome across families.

Among many others, these studies have provided mixed evidence for linkage and association of alcohol dependence to Chromosome 1 (Dick et al., 2002; Lappalainen et al., 2004), Chromosome 4 (Prescott et al., 2006), Chromosome 5 (Ehlers & Wilhelmsen, 2005), Chromosome 15 (Dick et al., 2004); the μ-opioid receptor gene (Nishizawa et al., 2006; Oslin et al., 2003), GABA receptor genes (Edenberg et al., 2004; Fehr et al., 2006; Sander et al., 1999), the serotonin transporter gene (LeMarquand et al., 1994; Sander et al., 1997), the dopamine receptor gene (Franke et al., 1999; Köhnke et al., 2005), and the ADH and ALDH2 genes (Crabb, Matusmoto, Chang, & You, 2003; Luczak, Glatt, & Wall, 2006)[2]. However, much of this research has yielded inconsistent results across groups of varying sample size, ethnicity, and clinical diagnoses. Furthermore, given the exhaustive list of proposed genetic markers, restricting the genetic transmission of alcohol abuse/dependence within families remains indistinct. However, those studies focusing on the genetic alteration of ethanol (e.g., ADH and ALDH2) have shown the greatest promise. Nonetheless, despite some ambiguity in the results, the findings from human genome studies support the suggestion that genes operate as a mechanism for the interfamilial transference of alcohol problems, such that similarities in genetic makeup between parents and offspring contribute to similarities in alcohol behavior.

3.2. Endophenotypes

Existing literature suggests that an endophenotype for a disorder should be heritable if there is a direct relationship between it and other susceptibility genes for the disorder (Carlson, Iacono, & McGue, 2004; van Beijsterveldt, van Baal, Molenaar, Boomsma, & de Geus, 2001; Yoon, Iacono, Malone, & McGue, 2006). One genetically influenced phenotype that has been associated with increased risk for alcoholism is the reduction of P300 (P3) amplitude during event-related potential (ERP) recordings. The P3 is a brain potential which indicates the amount of attentional resources required for encoding new information in working memory (van Beijsterveldt et al., 2001). The reduction in this P3 wavelength has shown to be more similar in MZ than DZ alcoholic twins (2004, Carlson et al., 2002), however heritability estimates appear to vary across gender, with percentages around 65% for boys, and 35% for girls, who were also substantially affected by shared environment (Yoon et al., 2006). A parental transfer risk has also been found with children of alcoholic parents exhibiting reduced P3 amplitude, however this parental risk effect appears greater in high-risk families, compared to low-risk families who were less likely to be exposed to the negative effects of alcohol or other substance abuse (Hill, Steinhauer, Lowers, & Locke, 1995; Hill, Yuan, & Locke, 1999).

Another genetically influenced characteristic associated with the increased risk for AUDs is the level of response (LR) to alcohol (Schuckit & Smith, 2000; Schuckit, Smith, Anderson, & Brown, 2004). This theory proposes that individuals with a low LR will require larger amounts of alcohol to produce the same effect (i.e., level of intoxication) that others experience with lower quantities. In fact, an early development of the need for increased levels of alcohol to produce the required effects (i.e. a low LR) has been shown to predict future alcohol abuse and dependence 10 to 15 years later (Schuckit & Smith, 2000, 2004). Analogous to family and twin studies, the heritability of LR aspects is estimated to be approximately 40% to 60% with genome-wide searches revealing significant correlations between 0.20 and 0.40 among first-degree relatives, but only 0.05 or less for unrelated individuals (Schuckit, Wilhelmsen, et al., 2005, Schuckit et al., 2001; Wilhelmsen et al., 2003). Furthermore, a 20-year cross-generational study of 40 father–offspring pairs revealed similarities in LR across two generations, with lower LR values found in offspring with a family history of alcohol dependence (Schuckit, Smith, Kalmijn, & Danko, 2005). The predictive ability of LR has also been revealed for both sons and daughters of alcohol dependent probands, with similarities in LR indices for both genders (Eng, Schuckit, & Smith, 2005; Schuckit et al., 2000).

3.3. Summary and critique

A plethora of research has provided mixed evidence for a specific gene, or genes, to explain the mechanism of the intergenerational transference of alcohol abuse or dependence. Similarly, whereas the identification of observable endophenotypes has provided similar heritably estimates as family studies, it is argued that given the polygenic nature of most genetic influences, it is not possible to

[2] Animal studies have identified similar chromosomal regions, however only human studies are referred to here.

implicate a specific gene, or gene combination, that contributes to the vulnerability toward AUDs (Walters, 2002). Nevertheless, these studies provide some evidence that the role of genetics is a likely contributor to the transference of alcohol behavior within families. It is argued however that the impact of biological influences can only be understood when evaluated in the context of environmental contributors, given that these factors have also shown to contribute to a family history of alcoholism. In other words, the interaction of genetic and environmental influences may increase the risk of children of parents with AUDs developing similar disorders under certain family environmental circumstances.

4. Genotype–environmental theories

Genotype × environment (G × E) research that has assessed lifetime prevalence of alcohol dependence has noted that individuals at high genetic risk for alcoholism are usually also exposed to high-risk environments (Heath & Nelson, 2002). As such, the intergenerational mechanisms that occur remain ambiguous such that the increased rates of alcoholism observed in children of problem drinkers does not distinguish whether it is the genetic transference from parents to offspring, poor family functioning due to parental problem drinking, or a combination of both genetic and environmental influences that contributes to the increase in risk of offspring alcohol misuse (McGue, 1997). To explore this, studies assessing the intergenerational effects of alcohol use behavior often discriminate between those environmental factors that are shared by siblings (SE or *c*), which includes parental and family influences, and non-shared environmental factors (NSE or *e*) which are unique to siblings (e.g., peer groups), and the genetic component shared by parents, offspring, and siblings (G or *a*). The majority of research into the transference of alcohol problems within families normally focuses on the first and last of these factors in determining the contribution of genetic and environmental influences.

Estimates of the percentage of variance in alcohol use due to shared environment vary excessively among the literature. For example, Koopmans and colleagues (e.g., Koopmans & Boomsma, 1996; Koopmans et al., 1999) have reported shared environment estimates of 37% to 54% among Dutch adolescent and young adults, whereas Maes et al. (1999) have provided estimates between 53% and 71% in a Virginian twin sample. Many G × E studies have found however that common environment interacts with heritability estimates according to age, zygosity, and gender (Han et al., 1999; Maes et al., 1999; Rose et al., 2001). Alternatively however, Prescott and Kendler (1999) revealed that in a twin study of 3516 MZ and DZ male twin pairs, common or shared environment only accounted for 3% to 11% of the variation in the development of alcohol abuse and dependence, with no major G × E effects. Furthermore, individual differences in these alcohol disorders were found to arise from an interaction between shared genes, and environmental influences not shared by family members. Similar shared environment rates have been observed by Whitfield et al. (2004) with shared environment contributing only 1% to 10% to alcohol intake. However, Heath et al. (1997) reported much lower rates with shared environment contributions of only 1% to 3%, and no G × E interactions, after including unlike-sex relative pairs in addition to same-sex relative pairs in their analyses. In stark contrast however, before and after controlling for the effects of sociodemographic and psychiatric predictors on the risk of alcohol dependence, Knopik et al. (2004) found that shared environment did not provide any variance to the disorder. They conjectured though that this finding did not exclude possible G × E interactions given that the interaction between genetic and shared environmental effects contribute to similarities of biological siblings reared together, but not to unrelated siblings reared together, or to biological siblings reared apart (Heath & Nelson, 2002).

It would appear by these results that, as a mechanism, shared environmental factors add little to the explanation of the intergenerational transference of alcohol problems, in some studies leaving over 90% of the variance in alcohol transference in families unaccounted for. Additionally, it would appear that G × E interactions appear greater for unshared environmental factors, suggesting that indirect effects of parental alcoholism act as a mechanism for the interfamilial transmission of alcohol disorders.

4.1. Family dysfunction

Considerable empirical evidence suggests that alcoholism adversely affects both the global family environment (Sher, Gershuny, Peterson, & Raskin, 1997; Velleman, 1992) and the psychological well-being of the alcoholic's offspring (Bijttebier & Goethals, 2006; Ellis, Zucker, & Fitzgerald, 1997; Hill, Lowers, Locke, Snidman, & Kagan, 1999; Rangarajan & Kelly, 2006). These difficulties with both family and personal functioning have resulted in children of alcoholics being recognised as an at-risk population for alcohol abuse, either directly as a means of coping, or indirectly through the association with deviant and substance using peers due to low parental monitoring (Serbin & Karp, 2004; Velleman, 1992).

Ellis et al. (1997) propose that children at the highest risk of developing AUDs are characterised by an aggregation of a number of alcohol specific (i.e., explicitly predicts alcohol abuse/dependence) and non-specific (i.e., increases the risk of developing alcohol abuse/dependence) factors occurring within the family. For example, co-occurring risks for the child include severe alcohol dependence and co-morbid psychopathology in both parents, high family violence, and parental modelling of alcohol use as a means of coping (Ellis et al., 1997).

The process of transference of alcohol problems from parent to child due to family dysfunction remains somewhat unclear however, given that negative family processes may either be the cause or the result of parental alcohol misuse. For instance, Barnow, Schuckit, Lucht, John, and Freyberger (2002) have found a family history of alcoholism to be unrelated to alcohol problems in a group of German adolescents. However, the parental co-occurring psychiatric diagnoses, and the offspring's perceived parental rejection was related to the child's aggression/delinquency, which in turn led to association with substance using peers and thus, to alcohol problems. When those children diagnosed with conduct disorder and/or antisocial personality disorder were removed from the analyses however, aggression/delinquency was no longer associated with peer group substance use, and therefore no longer acted as a predictor for alcohol use problems. Family psychopathology however remained a significant predictor of alcohol problems suggesting that this factor increases the risk for a range of behavioral and emotional problems in children of alcoholics.

In contrast, an investigation of paternal alcohol consumption, family stressors, and adolescent AUDs revealed that a family history of alcoholism increased the risk of offspring AUDs twofold (Sher et al., 1997). Furthermore, Sher et al., revealed that although the presence of an alcoholic father was significantly associated with several childhood stressors such as verbal, physical, emotional and sexual abuse, and that several of these stressors were associated with a lifetime AUD diagnosis, these childhood stressors only partially mediated the relationship between parental and offspring AUDs.

Other research has found some inverse relationships between parental and child alcohol use (Harburg et al., 1990; Van Gundy, 2002; Webster et al., 1989). Van Gundy's study of Russian family alcohol use behavior, and family interaction and conflict, found that father's verbal abuse of mothers significantly increased offspring drinking, however father's alcohol-related violence *decreased* the child's drinking (Van Gundy, 2002). Harburg et al. (1990) explain this latter finding as the "fall-off" or "aversive transmission" effect where offspring of problem drinkers moderate their own drinking due to their perceptions of their parent's problematic drinking behavior.

It should be noted however that Menees and Segrin (2000) have found no difference in the specificity of problematic family environments

between children of alcoholics and other children exposed to other family stressors such as parental divorce, death, or major illness. This argues against the notion that parental alcoholism is a sufficient cause for family dysfunction, and that unstable family environments appear to be equally prevalent among families exposed to other significant stressors. Irrespective, it appears that a dysfunctional family environment operates as a partial mechanism for the intergenerational transference of alcohol problems, however what remains unclear is whether this process operates directly or indirectly.

4.2. Summary and critique

As shown, a large amount of literature has addressed the relationship between genetic and environmental factors in describing the mechanism for the intergenerational transference of alcohol problems, yet findings remain inconclusive. Specifically, genetic and environment studies have provided estimates from 0% to 71% for shared environment, and inconsistencies remain as to the genetic × environment interaction. Furthermore, the discrepancies in the findings assessing the relationship between varied elements of family dysfunction and the interfamilial transference of drinking problems seem to suggest that AUDs in the offspring of alcohol abusing parents may operate through other influences resulting from parental alcohol misuse.

Overall, the range of biosocial theories have provided some evidence (albeit mixed) for heritable and milieu related influences in the intergenerational transference of alcohol problems. However, this literature leaves a large proportion of the variance in this process unaccounted for. Further, studies show that certain children do not develop alcohol use problems despite having an alcoholic parent, whereas others acquire such disorders regardless of non-alcoholic parents. It is therefore suggested that other mechanisms are operating which account for a part of the variance in interfamilial alcohol problems, which may serve as either protective or risk factors. Particularly, it is proposed that specific social learning cognitions regarding the anticipation, expectancy, memory, and modelling of alcohol use are fundamental in determining alcohol use behavior (Oei & Baldwin, 1994; Oei & Morawska, 2004).

5. Cognitive perspectives

The role of social factors cannot be overlooked as significant antecedents in the initiation of problem drinking. However, certain social constructs (e.g., social norms and gender identification) need to be considered simultaneously with the cognitive aspects associated with them and their interactive effects on the intergenerational transference of alcohol consumption. Specifically, children develop early expectations about gender through observation of parents and then form schemas that influence how they perceive the behaviors of men and women, and in turn, how these behaviors adhere to social norms such as appropriate alcohol use behavior.

Given that gender plays a fundamental role in the formation of identity (and social norms), association with either masculine or feminine traits may be a significant means through which distinctive gender-related styles of alcohol use develop (Wilsnack, Vogeltanz, Wilsnack, & Harris, 2000). For instance, males typically display a considerably higher prevalence of drug and alcohol-related problems than women, and identification with the masculine role such as dominance, assertiveness, and independence may contribute to this (Horwitz & White, 1987). Alternatively, exaggerations of conventional female stereotypes may encourage females to be submissive, helpless and dependent thus increasing the risk of alcohol misuse (Horwitz & White, 1987). It should be noted however that convergence in gender behaviors and changes in societal norms related to gender identity has led to similarities in alcohol use quantities in both males and females. This would indicate that the changes in perceptions of gender role differentiation may also be mirrored in changes of alcohol use behavior. As such, the motivations to drink (e.g., coping with negative emotions, stress control, social facilitation, adherence to perceived societal norms) may be different in each sex but underlie the transference of cognitions associated with typical gender roles from parents to children.

The increase in the number of young adolescents and adults drinking alcohol at risky levels (Chikritzhs et al., 2003) suggests that antecedent characteristics may exist prior to the individual's first experience with alcohol. Children as young as six years old have shown to possess some understanding of the contextual, motivational, and normative aspects of alcohol consumption and behavior (Miller, Smith, & Goldman, 1990; Oei & Angel, 2005; Zucker, Kincaid, Fitzgerald, & Bingham, 1995). Based on these findings, it has been argued that a large proportion of intra-family alcohol use behaviors may occur through the transference of alcohol cognitions. The results by Nash, McQueen, and Bray (2005) seem to support this possibility. Their results indicate that adolescent's perception of their parent's approval or disapproval of their alcohol consumption was related to the child's drinking habits, such that perceived parental disapproval was associated with increased self-efficacy for refusing alcohol, and lower alcohol consumption. Similarly, Zhang, Welte, and Wieczorek (1997) found that parental attitudes towards alcohol exerted a greater influence on younger adolescents' alcohol use than did parental alcohol use behavior.

Zucker et al. (1995) have proposed however that information about alcohol is developed much earlier than adolescence, and suggests that cognitive schemas are used by very young children to organize their knowledge and beliefs about alcohol, and arise from parental alcohol use. Their results revealed that most children could identify alcohol type by photographs by the age of three, and that these children held two common alcohol schemas also held by the greater culture; 1) that alcohol consumption is done more by adults than children, and 2) that alcohol consumption is done more by males than females. Similarly, Ouellette, Gerrard, Frederick, Gibbons, and Reis-Bergan (1999) have argued that prior to alcohol use children develop an image of a 'typical' drinker through levels of exposure, and then perceive themselves as similar or different to that prototype. It is then suggested that the child's identification with, and favourability of, that image is related to the child's willingness to drink, and the likelihood of their consuming alcohol should the opportunity arise.

5.1. Alcohol expectancies and drinking refusal self-efficacy

Another cognitive theory for describing the relationship between alcohol cognitions and drinking behavior in the same individual is the Alcohol Expectancy Theory (Oei & Baldwin, 1994). However, it is argued that this model can be extended to explain the relationship between parental drinking related variables and offspring alcohol consumption. For instance, this model examines the relationship between alcohol expectancies (AE) and drinking refusal self-efficacy (DRSE), which are beliefs about specific outcomes and self-control behavior associated with alcohol consumption. The theory proposes a two-process model of alcohol use and abuse; 1) an acquisition phase, based on instrumental learning or modelling processes in which alcohol expectancies are formed, and 2) a maintenance phase based on classical conditioning, in which unconscious conditioned processes automatically initiates a drinking response. In terms of an interfamilial transference example, expectancies as to the effect of alcohol originate as a result of parental modelling and once these cognitions become established, they guide the child's behavior when exposed to alcohol. Once consumed, these expectancies are reinforced, thus leading to the maintenance of alcohol use behavior. It is suggested that alcohol expectancies are important in evaluating the decision of whether or not to drink, whereas drinking refusal self-efficacy arbitrates the behavioral response. These constructs have been discussed in previously published research (Lee & Oei, 1993; Oei & Baldwin, 1994; Oei & Burrow, 2000; Oei,

Hasking, & Young, 2005; Young, Oei, & Crook, 1991) and will therefore only be discussed briefly here.

Research suggesting that the overall effects of alcohol operates through the interaction of its physiological effects, and the beliefs that an individual holds regarding these effects, forms the basis for the concept of alcohol expectancies (AEs). AEs are generally expressed in the form of contingencies, or 'if…then' statements (e.g., If I drink alcohol...then I will be happy/sad/depressed). These expectancies predetermine the individual's choice to use (or not use) alcohol and also their subsequent behavior, which is driven by the anticipation of the effects that alcohol will have, such as increased sociability or tension reduction (Oei & Morawska, 2004). Successive confirmation of these expectancies can reinforce drinking behavior and, in the case of optimistic outcomes, place the individual at risk of persistent problem drinking. The predictive ability of global, positive, social, and physical AEs in alcohol misuse has been shown to contribute 10%–19% of variance in current alcohol use (Leigh, 1989) whereas other studies have revealed that positive and negative expectancies explained between 51% and 54% of the variance in alcohol use in a university student population (Brown, Goldman, Inn, & Anderson, 1980; Leigh & Stacy, 1993).

In contrast, drinking refusal self-efficacy (DRSE) has been defined as the perceived ability to refuse alcohol in a specific situation (Lee & Oei, 1993; Oei & Burrow, 2000; Young et al., 1991), rather than whether or not one chooses to drink. Unlike AE, which only predicts frequency, DRSE determines both the frequency and level of alcohol consumption (Oei & Morawska, 2004), such that when given the opportunity to drink, individuals with low DRSE tend to consume larger amounts of alcohol more frequently (Hasking & Oei, 2002; Lee & Oei, 1993). It has been shown that DRSE scores can predict alcohol consumption levels, discriminate between problem and non-problem drinkers, and predict treatment responses in adults from general and clinical populations (Annis, 1990; Baldwin, Oei, & Young, 1993; Lee & Oei, 1993; Young et al., 1991). Findings from previous research would suggest that the constructs of AE and DRSE form the foundations of the transfer of alcohol use behavior between generations, and has repeatedly been demonstrated in alcohol research.

5.2. The intergenerational transference of alcohol cognitions

It has been suggested that children with a family history of alcohol abuse differ from those without a family history of alcohol abuse in terms of their expectations regarding alcohol's effects. Findings by Brown, Creamer, and Stetson (1987) and Sher et al. (1991, 2005) revealed that AEs differed between offspring of alcoholic parents and offspring from non-alcoholic families, such that those adolescents with a family history of alcohol misuse expected more enhanced cognitive and motor abilities. Furthermore, although not central to their research, their pattern of results also revealed that non-alcohol abusing adolescents with alcoholic parents had a higher number of positive expectations to alcohol use than non-alcohol abusing children from non-alcoholic families. This would imply that these children adopt their parent's expectancies regarding the perceived advantageous effects of alcohol, without the influence of their own alcohol experiences. Brown et al. (1987) proposed that children may acquire the expectation of enhanced functioning via their alcohol abusing parent's self-report of improved performance whilst drinking, or via the child's observation of the parent's reduction in withdrawal symptoms, once alcohol consumption is resumed.

This argument has been supported recently by Shen, Locke-Wellman, and Hill (2001), who found that offspring at high risk of developing alcoholism (i.e., presence of alcoholic father and uncle) held beliefs about the effects of alcohol that were similar to their parents, compared to low-risk controls. Furthermore, high-risk adolescents were found to possess higher expectations that alcohol would provide enhanced social facilitation, compared to their low-risk counterparts. However, no between group differences were observed between high and low-risk adolescents at a 3-year follow-up. It should be noted however that at the first assessment, only 12% of offspring (high and low-risk combined) were consuming alcohol regularly, whereas 51% were drinking regularly at the time of the second assessment. When Shen et al., compared drinkers to non-drinkers, those adolescents who had begun drinking reported higher scores for social functioning expectancies compared to non-drinkers. This finding supports those by Smith, Goldman, Greenbaum, and Christiansen (1995) who revealed that the consumption of alcohol induced a positive rather than a negative feedback loop on alcohol expectancies, such that the more positive expectancies for the use of alcohol, the greater the level of drinking, which in turn reinforced further positive alcohol expectancies.

The formation of these alcohol cognitions prior to drinking experience indicates that the knowledge that forms these beliefs is derived from sources other than actual drinking behavior. For children, arguably the most accessible and significant models displaying alcohol-related behaviors would be their parents. It has been suggested in previous literature that exposure to parental behavior influences the child's behavior directly through imitation and modelling (e.g., Bandura, 1977), however adoption studies on alcohol use transference suggest that risk for intergenerational transmission is elevated even in the absence of contact with the alcoholic parent, making the argument for observation as a requirement for transmission debatable. Given this premise, the following hypothesised model will propose an existing relationship between offspring's alcohol cognitions and subsequent drinking behavior, via their parent's alcohol cognitions and their alcohol use, rather than directly from parent to child behavior.

5.3. A cognitive model of the intergenerational transference of alcohol use behavior

The full cognitive model is shown in Fig. 1 encompassing a behavioral component (broken lines) and a cognitive component (bold lines). The behavioral element assesses the relationship between parental alcohol behavior and child cognitions, and suggests that parental behavior indirectly influences their offspring's behavior through the child's beliefs and expectancies, rather than directly from parent to child behavior. In contrast, the proposed cognitive component

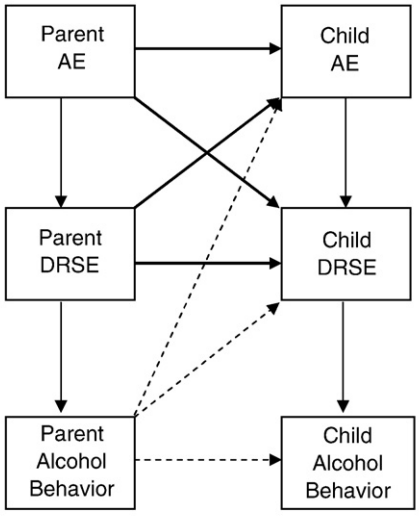

Fig. 1. The full cognitive model of the intergenerational transference of alcohol problems, encompassing a behavioral component (broken lines) and a cognitive component (bold lines).

indicates that it is the parent's alcohol cognitions that indirectly influence their offspring's alcohol use via their alcohol cognitions, and suggesting that parents and children share cognitions regarding the effects of alcohol.

Although there has been minimal research of the possible relationships between parent and child AE and DRSE, there are a number of theoretical reasons to suggest that this proposed relationship exists between parental alcohol expectancies and drinking self-efficacy beliefs, and the same in their offspring. Firstly, it appears that the observation of parental drinking contributes to the child's perceptions and expectations of alcohol's effects, which in turn leads to the child's future alcohol use behavior, rather than just through mimicry of parental models (Brown, Tate, Vik, Haas, & Aarons, 1999; Brown et al., 1987; Erblich, Earleywine, & Erblich, 2001; Oei & Baldwin, 1994; Ouellette et al., 1999; Zucker et al., 1995). Whereas only a small number of studies have assessed the differences in alcohol expectancies between children of alcoholics and non-alcoholics, results indicate that the offspring of problem drinkers report more positive expectations to the effects of alcohol, compared to their non-alcoholic family counterparts (Brown et al., 1987; Lundahl, Davis, Adesso, & Lukas, 1997), however this pattern was not found in a sample of adult offspring (Finn, Sharkansky, Brandt, & Turcotte, 2000; Ohannessian & Hesselbrock, 2004).

Secondly, it has been shown that a positive association between alcohol expectancies and problem drinking was greater for individuals with a family history of problem drinking, compared to those without such history, and that the influence of alcohol expectancies on problem drinking increased with the number of relatives in the family with alcoholism (Conway et al., 2003). As proposed by Brown et al. (1987), it seems plausible to suggest that parent's alcohol cognitions could be transferred to their offspring via verbal affirmations of alcohol's perceived benefits (e.g., "alcohol is good for relaxing after a hard day's work"). Moreover, as previously mentioned, Shen et al. (2001) (see also, Johnson, Nagoshi, Danko, Honbo, & Chau, 1990) revealed a number of significant positive parent/child correlations for alcohol expectancies, indicating that high-risk offspring held similar beliefs about the effects of alcohol as their parents, which remained relatively unchanged with age and alcohol experience. Thus, the transmission of a particular drinking style from parent to child may be better conceptualised as acquiring a set of beliefs, or more precisely, expectancies, about the outcomes of drinking alcohol, as well as beliefs about their ability to refuse alcohol in a particular situation.

Whereas a number of studies have shown that parental alcohol behavior and attitudes can predict their offspring's alcohol use, no study to date has directly assessed parents' alcohol expectancies and drinking refusal self-efficacy with those of their children's, in the prediction of offspring alcohol use. Previous research has supported the importance of parental and child cognitions and behavior in interfamilial transference, however, most studies have considered these constructs in isolation. Forthcoming research will assess the ability of the proposed model in predicting this intergenerational transmission of alcohol problems within families.

5.4. Concluding comments

The consequences of alcohol misuse, especially in adolescents and young adults, are evident, and therefore indicate the importance of prevention and intervention strategies. It is clear from the alcohol transference literature that there exists a strong relationship between parental and offspring alcohol abuse. Twin and adoption studies have suggested that these similarities may be due to a genetic component shared by first-degree relatives, yet many have argued that the role of genetics can only be understood when environmental influences are considered given that these factors also contribute to the variability in problem drinking behaviors. That is, we know from family studies that shared and non-shared environmental similarities can interact with genetic similarities, creating a stronger effect compared to their individual influences. However, research has shown that this theory is not sufficient in explaining a large proportion of the variance in the transference of alcohol problems within families.

More recent studies have implied that psychological factors such as the transference of beliefs, attitudes, and perceptions from parent to offspring are important in the development of alcohol problems. Nonetheless, to date, this has not been well articulated in the literature, and it is therefore argued that the addition of a cognitive model for the intergenerational transference of alcohol problems is somewhat timely, and has many advantages. Most specifically, the implications of the cognitive model include the provision of information relevant to prevention and treatment, given that cognitions are particularly modifiable to change. Such preventative measures would adopt a proactive rather than reactive stance on alcohol misuse in children and young adults whereby maladaptive cognitions as to the perceived benefits of excessive alcohol use could be altered or avoided very early. Similarly, awareness of the cognitive mechanisms driving alcohol use in a young population enables preventative campaigns to employ more specific and effective messages by educating parents as to their role in the transference of alcohol messages to their offspring.

Theoretically, whereas previous research has assessed the importance of alcohol expectancies (Brown et al., 1987, 1999; Conway et al., 2003) and self-efficacy (Bandura, 1982, 1999) in determining alcohol use behavior, most studies have considered these in isolation, or have not considered the transference of such cognitions between generations. Furthermore, such a testable model adds richness to the conceptualisation of the mechanisms for the transference of alcohol problems within families. Specifically, the opportunity arises to incorporate cognitive influences into the intergenerational literature, resulting in a more thorough genetic × environment × cognitive theory.

In sum, the current review has aimed to provide an understanding of the possible mechanisms responsible for the perpetuation of alcohol problems in families, and has proposed a model indicating the significant role of parental alcohol cognitions on their child's alcohol cognitions, and subsequent alcohol use behavior. Literature assessing the intergenerational transference of gambling (Oei & Raylu, 2004) indicates that such a cognitive model has merit. For example, Oei and Raylu proposed a similar model that revealed parental gambling cognitions influence their offspring's gambling behaviors, via offspring cognitions, whereas parent's gambling behaviors influenced offspring gambling behavior directly. It is anticipated that the focus on the familial transfer of alcohol expectancies and drinking refusal self-efficacy cognitions within the current model, and the modifiable nature of these variables, will provide a theoretical basis for the treatment of alcohol use disorders.

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