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Variation of types of alcoholism: Review and subtypes identified in Han Chinese

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ABSTRACT

Alcoholism, as it has been hypothesized, is caused by a highly heterogeneous genetic load. Since 1960, many reports have used the bio-psycho-social approach to subtype alcoholism; however, no subtypes have been genetically validated. We reviewed and compared the major single-gene, multiple-gene, and gene-to-gene interaction studies on alcoholism published during the past quarter-century, including many recent studies that have made contributions to the subtyping of alcoholism. Four subtypes of alcoholism have been reported: [1] pure alcoholism, [2] anxiety/depression alcoholism, [3] antisocial alcoholism, and [4] mixed alcoholism. Most of the important studies focused on three genes: *DRD2*, *MAOA*, and *ALDH2*. Therefore, our review focuses on these three genes.
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1. Introduction

Some family studies (e.g., Merikangas, 1990) have shown that genetics is a major factor in alcoholism. Others (Ferguson and Goldberg, 1997; Reich et al., 1999) have suggested that the heritability of alcoholism is at least 50%. Alcoholism is three to five times more frequent in the parents, siblings, and children of alcoholics than in the general population (Cotton, 1979). Twin studies have shown the heritability of alcoholism by comparing concordance rates. Pickens et al. (1991) report a significant difference between monozygotic and dizygotic twin concordance for alcohol dependency in 886 male same-sex twin pairs. An investigation (Kendler et al., 1992) of 1030 female–female twin pairs in Virginia found a significant genetic component for alcoholism. Moreover, a Danish study (Goodwin et al., 1973) of 133 male adoptees separated from their parents by 6 weeks of age found that 18% of those with a biological father who was an alcoholic developed alcoholism compared with only 5% of the adoptees who did not have a biological father with a history of alcoholism. Taking all these findings together suggest a genetic component in alcoholism.

Abbreviations: ALC, alcoholism; ALDH2, aldehyde dehydrogenase 2; Antisocial ALC, antisocial alcoholism; Antisocial non-ALC, antisocial non-alcoholism; ANX/DEP ALC, anxiety/depression alcoholism; ASPD, antisocial personality disorder; DOPAC, 3,4-dihydroxyphenylacetic acid; DOPAL, 3,4-dihydroxyphenyl-acetaldehyde; DRD2, dopamine D2 receptor; HA, harm avoidance; MAOA, monoamine oxidase A; Mixed ALC, mixed alcoholism; MTHFR, methylenetetrahydrofolate reductase; NS, novelty seeking; Pure ALC, pure alcoholism.

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Many candidate genes that contribute to alcohol dependence susceptibility have been proposed, yet few of the published results leading to the proposal of these candidate genes can be replicated (Dick and Foroud, 2003; Reich et al., 1999). Being a multi-factorial mental disorder, alcoholism is also influenced by heterogeneity and socio-cultural factors (Cloninger, 1987; Merikangas, 1990). The typology of alcoholism to reduce heterogeneity is therefore important and critical in both the area of research and clinical practice. However, exploration of genetic underpining after subtyping of alcoholism is largely overlooked.

2. Review of alcohol subtypes

The clinical heterogeneity of alcoholism may be related to genetic heterogeneity (Cloninger, 1987). The typology of alcoholism is, therefore, important for reducing inconsistencies both in research and in clinical practice. Individual subtypes of alcoholism may highlight genetic perturbations within particular domains of psychopathology that might not be revealed when studying alcoholism as a whole. Therefore, differentiating subtypes of alcoholism may reduce contradictory factors and uncover a more powerful association between genes and specific subtypes of alcoholism.

Since 1960, researchers have used a bio-psycho-social approach to subtype alcoholism, which assumes that the many differences between alcoholics are contributed by biological, psychological, and socio-cultural factors. Jellinek et al. (1953) published the first comprehensive analysis of alcoholism in the twentieth century. There are histories of medical treatments for alcoholism in America from the middle of the 18th century. Lesch et al. (1988) divided alcoholism into 4 subtypes in their comprehensive analysis of alcoholism.

Twenty-six years later, Morey and Skinner (1986) classified alcoholics into three subtypes: [1] early-stage problem drinkers: had drinking problems in their early stage but did not develop symptoms of severe alcohol dependence; [2] affiliative drinkers: more likely to drink socially, and often drink every day, which causes moderate alcohol dependence; [3] schizoid drinkers: more socially isolated and withdrawn from social activities, often drink alone, and severely alcohol dependent.

Zucker et al. (1987) classified alcoholics into four subtypes: [1] antisocial alcoholics: had alcoholic problems and antisocial behaviors at a very young age, which might be an inherited vulnerability and cause a poor prognosis; [2] developmentally cumulative alcoholics: drink because of cultural factors and gradually become alcoholics; [3] negative-affect alcoholics: more often females who drink for social relationships or to eliminate their negative emotions; [4] developmentally limited alcoholics: patients in their early stage more often drink heavily in the early stage and gradually control themselves to drink only at social activities in order to do allow them to fulfill their responsibilities and social roles.

Lesch et al. (1988) described four types of alcoholics with different reasons for developing an addiction, different withdrawal syndromes, and different prognoses, as well as those who may benefit from different therapeutic approaches. Type I alcoholics show limited social and psychological pathology: less than with total abstinence but no marked craving for alcohol and they feel healthy in their psychosocial situation. They may develop a strong and immediate craving whenever they consume even a small amount of alcohol. Type II alcoholics use alcohol for self-medication and conflict solving. Without social, psychological, or somatic disturbances, patients of this subtype normally use only a small amount of alcohol. They appear to have passive lifestyle most of the time but are usually over-adapted to society. Type III alcoholics may exhibit psychic, somatic, and social problems after drinking. They often have comorbid affective disorders and a positive family history of both alcohol dependence and affective disorders. Type IV alcoholics have demonstrable psychic, somatic, and social behavior problems. They might have had brain damage during childhood, a dysfunctional family or no family at all, and no supportive social life, which led to behavioral problems during childhood.

Cloninger (1987) proposed a neurobiological learning model of alcoholism that described two genetic subtypes: Type I (milieu-limited) and Type II (male-limited) alcoholism. Type I alcohol use disorders include late-onset drinking behavior, more psychological dependence, high harm avoidance (HA), and low novelty seeking (NS), whereas Type II alcohol use disorders include early-onset drinking behavior, more behavioral disturbances, low harm avoidance, and high novelty seeking. Each of the personality dimensions is postulated to be associated with a particular neurotransmitter system. Specifically, NS is mediated by the dopaminergic system and HA by the serotoninergic system (Cloninger, 1987). Evidence supports the hypothesis that dopaminergic and serotonergic neurotransmission interact at the molecular level (Kapur and Macdonald, 1996; Prisco et al., 1994). Associations between NS and serotonin (5-HT) and between HA and dopamine (DA) have also been reported (Kremer et al., 2005; Tomer and Aharon-Peretz, 2004) (Table 1).

Five years later, Babor et al. (1992) used an empirical clustering technique to identify two types of alcoholism across 17 defining characteristics. Type A alcoholics are characterized by late onset, fewer childhood risk factors, less severe dependency, fewer alcohol-related problems, and less psychopathological dysfunction. Type B alcoholics are characterized by childhood risk factors, family alcoholism, early onset of alcohol-related problems, greater severity of dependency, polydrug use, a more chronic treatment history, greater psychopathological dysfunction, and more life stress.

Studies that have tried to genetically validate different subtypes of alcoholism have yielded only limited findings. The methylenetetrahydrofolate reductase (*MTHFR*) polymorphism has been associated with Lesch Type III alcoholism, with depression, and with Babor Type A alcoholism (Benyamina et al., 2009). However, no subtypes have been completely genetically validated. In addition, these subtypes may be difficult to use clinically because of cultural differences. Most patients diagnosed with alcohol dependence meet most of the DSM-IV-TR diagnostic criteria, which suggests that the disorder is homogeneous. However, alcoholic patients frequently have other comorbid mental illnesses. Using the comorbidity of other mental illnesses to reclassify the subtypes of alcoholism may be worth investigating.

3. Subtypes of alcoholism identified in Han Chinese

We previously categorized alcoholism (ALC) into four subtypes (Huang et al., 2008; Lu et al., 2012): [1] pure alcoholism (Pure ALC), [2] anxiety/depression alcoholism (ANX/DEP ALC), [3] antisocial alcoholism (Antisocial ALC), and [4] mixed alcoholism (Mixed ALC).

Table 1
Genetic associations with different subtypes of alcoholism (Huang et al., 2004, 2007; Lee et al., 2009, 2010; Lu et al., 2002, 2003, 2005, 2012; Wang et al., 2007).

Gene	Subtype of alcoholism
Single gene	
DRD2 TaqIA polymorphism	Anxiety/depression alcoholism
MAOA-uVNTR polymorphism	Pure alcoholism
ALDH2 polymorphism	Anxiety/depression alcoholism
	Pure alcoholism
	Mixed alcoholism
Gene-to-gene interaction	
DRD2 TaqIA polymorphism	Anxiety/depression alcoholism
Stratified by *MAOA-uVNTR* 3-repeat	
DRD2 TaqIA polymorphism	Antisocial alcoholism
Stratified by *MAOA-uVNTR* 4-repeat	
DRD2 TaqIA polymorphism	Anxiety/depression alcoholism
Stratified by *ALDH2* polymorphism	Antisocial alcoholism
	Antisocial non-alcoholism
Interaction of *ALDH2* and *DRD2 TaqIA*	Antisocial personality disorder
polymorphism	Antisocial non-alcoholism
	Anxiety/depression alcoholism
Interaction of *ALDH2* and *MAOA-uVNTR*	Antisocial alcoholism
polymorphism	Anxiety/depression alcoholism

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