Many epidemiological and clinical studies have shown that light-to-moderate alcohol (Alc) consumption is associated with reduced risk of coronary heart disease (CHD) and total mortality in middle-aged and elderly men and women. The plausible mechanisms for the putative cardioprotective effects include increased levels of high-density lipoprotein cholesterol, prevention of clot formation, reduced platelet aggregation, promotion of blood clot dissolution, and lowering of plasma lipoprotein (a) concentration. Individuals who need to be treated with lipid-lowering drugs, such as dyslipidemic or CHD patients, may benefit from these effects of Alc. Because hypolipidemic treatment is usually continued for life, an important issue is the suitability of Alc consumption in these patients. In the present review, the beneficial effects of Alc consumption on CHD risk, its side effects, and its safety and suitability when coadministered with hypolipidemic drugs are discussed.

Many epidemiological and clinical studies have shown that light-to-moderate alcohol (Alc) consumption is associated with reduced risk of coronary heart disease (CHD) and total mortality in the middle-aged and elderly of both genders (Rimm et al., 1999; Rehm et al., 2003). Men and women who consume a moderate amount of Alc, defined as one to four and one to two drinks, respectively, for 5 or 6 days per week had substantial reduction of major coronary events compared with nondrinkers [odds ratios: men 0.31 (95% confidence interval 0.22 to 0.45); women 0.33 (0.18 to 0.59)] (McElduff and Dobson, 1997). One thousand years ago, a “prescientific” observation of the beneficial effect of Alc was described by Hildegard of Bingen (1098–1179; Bingen, Germany), who applied a special “wine recipe” to treat cardiovascular disease (Böhm et al., 2004). The inverse alcohol-atherosclerosis association was also pointed out by pathologists early in the previous century. At the same time, there is no doubt that treatment with lipid-lowering drugs has decreased cardiovascular disease mortality (Collins et al., 2003; Harrington, 2004). Treatment with lipid-lowering drugs is recommended for lifetime, unless there are contraindications. The question is whether physicians can also recommend social Alc consumption to their patients while they are on lipid-lowering drug therapy. This review focuses on the issue of concomitant Alc consumption and lipid-lowering drug use.

**Alc and CHD**

The risk of death from all causes has been found to be significantly lower among men who drink moderately on a regular basis compared with abstainers (Rimm et al., 1999; Rehm et al., 2003). When data from various studies are combined, there seems to be a decline in the risk of myocardial infarction at doses of up to one drink per day, with little further change in this risk with increased Alc intake (Rimm et al., 1999). Makela et al. (1997) observed that light-to-moderate Alc consumption was associated with a 12–14% reduction in CHD deaths among Finish men aged 30 to 69 years. In the metanalysis of Rimm et al. (1999), a 30-g daily intake of Alc was associated with a 25% reduction in the risk of CHD. This benefit was attributed to lipid, lipoprotein, and fibrinogen alterations. The dose-response curve of Alc consumption in relation to total mortality and CHD risk is

**ABBREVIATIONS:** Alc, alcohol; CHD, coronary heart disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein; VLDL, very low-density lipoprotein; TG, triglyceride; AST, aspartate aminotransferase; Apo, apolipoprotein; NA, nicotinic acid.
usually found to be J- or U-shaped (Andreasson, 1998; Gaziano et al., 2000). The risk is higher in individuals not consuming any Alc than in cases of moderate consumption, with the lower risk observed at 20 g per day. This risk is increased in individuals that consume higher doses of Alc. When the average consumption per day rises to over 70 g, the risk becomes greater than that for abstainers (Rehm et al., 2003). The relationship with CHD mortality was found to be inverse or L-shaped with apparent risk reductions even in the highest category of ≥2 drinks per day (Gaziano et al., 2000). Moreover, the pattern of drinking seems to differentiate the effect of Alc on CHD. Whereas regular light-to-moderate drinking is beneficial for CHD risk, an irregular pattern of heavy drinking (≥5 drinks for women and ≥9 for men on one occasion) seems to be related to major coronary events (McElduff and Dobson, 1997), as well as other types of cardiovascular death, such as stroke and sudden cardiac death (Rehm et al., 2003). Finally, people who accompany Alc drinking with meals or snacks had a lower risk for CHD (Rehm et al., 2003).

**Biological Mechanisms Responsible for the Beneficial Effect of Alc**

It has been proposed that the protective effect of Alc is mediated through the increase of high-density lipoprotein (HDL) cholesterol, because its levels are inversely related to CHD (Agarwal, 2002). According to Rimm et al. (1999), consuming 30 g of Alc per day increases HDL cholesterol levels by 4 mg/dl (0.10 mM), which in turn is equivalent to an estimated 17% reduction in CHD risk. This Alc-induced increase in HDL cholesterol levels is attributed to the following mechanisms. First, Alc induces the hepatic synthesis of apolipoprotein (apo)AI and apoAII (the main components of HDL particles) (Rimm et al., 1999; Agarwal, 2002). It has been estimated that an average individual consuming 30 g of Alc per day would show an 8 mg/dl (0.20 mM) increase in the plasma concentration of apoAI (Rimm et al., 1999). ApoAI constitutes the precursor of HDL particle formation. Through its binding to ATP-binding cassette transporter A1, phospholipids attach to apoAI to form disk-like particles (pre-β-HDL) (Kolovou et al., 2006). Free cholesterol from peripheral cells then effluxes to pre-β-HDL that are transformed to mature-spherical HDL after cholesterol esterification (Kolovou et al., 2006). Second, Alc metabolism raises triglyceride (TG) concentration, which in turn induces the secretion of TG-rich lipoproteins by the liver, and increases the activity of TG lipase. Concomitantly, the lipolysis of TGs in lipoprotein particles [such as very-low-density lipoproteins (VLDL) and chylomicrons] increases the flow of cholesterol from these particles to HDL particles (Rimm et al., 1999). Third, Alc reduces cholesterol ester transfer protein activity, leading to reduced transfer of cholesteryl ester from the core of HDL to more atherogenic particles in exchange for TGs. Given that TG-rich HDL particles are prone to catabolism, Alc indirectly decreases the removal of circulating HDL cholesterol by the latter mechanism (Fumeron et al., 1995). In summary, Alc, by promoting the production of HDL constituents, by enhancing the flow of cholesterol to HDL particles, and by delaying their catabolism, favors raising HDL cholesterol concentration (Fig. 1). Early observations suggested that Alc mainly raised levels of HDL-3 particles, not HDL-2, but more recent studies have found that both subfractions are increased (Rimm et al., 1999).

Increased HDL cholesterol levels can partially explain the protective effect of alcoholic beverages. Other biological mechanisms underlying the protective effect of Alc include the increase of paraoxonase activity, the reduction of lipoprotein (a) levels, the inhibition of blood clotting and platelet aggregation, the improvement of insulin sensitivity, the lowering of plasma homocysteine levels, the estrogen levels increase, and the reduction of stress. Moreover, moderate alcohol intake has been reported to confer protection against

![Fig. 1. The mechanisms underlying the Alc-induced elevation of HDL cholesterol levels. I, nascent HDL formation through enhanced hepatic synthesis of apoAI and apoAII (the main components of HDL particles) (Rimm et al., 1999; Agarwal, 2002). It has been estimated that an average individual consuming 30 g of Alc per day would show an 8 mg/dl (0.20 mM) increase in the plasma concentration of apoAI (Rimm et al., 1999). ApoAI constitutes the precursor of HDL particle formation. Through its binding to ATP-binding cassette transporter A1, phospholipids attach to apoAI to form disk-like particles (pre-β-HDL) (Kolovou et al., 2006). Free cholesterol from peripheral cells then effluxes to pre-β-HDL that are transformed to mature-spherical HDL after cholesterol esterification (Kolovou et al., 2006). Second, Alc metabolism raises triglyceride (TG) concentration, which in turn induces the secretion of TG-rich lipoproteins by the liver, and increases the activity of TG lipase. Concomitantly, the lipolysis of TGs in lipoprotein particles [such as very-low-density lipoproteins (VLDL) and chylomicrons] increases the flow of cholesterol from these particles to HDL particles (Rimm et al., 1999). Third, Alc reduces cholesterol ester transfer protein activity, leading to reduced transfer of cholesteryl ester from the core of HDL to more atherogenic particles in exchange for TGs. Given that TG-rich HDL particles are prone to catabolism, Alc indirectly decreases the removal of circulating HDL cholesterol by the latter mechanism (Fumeron et al., 1995). In summary, Alc, by promoting the production of HDL constituents, by enhancing the flow of cholesterol to HDL particles, and by delaying their catabolism, favors raising HDL cholesterol concentration (Fig. 1). Early observations suggested that Alc mainly raised levels of HDL-3 particles, not HDL-2, but more recent studies have found that both subfractions are increased (Rimm et al., 1999). Increased HDL cholesterol levels can partially explain the protective effect of alcoholic beverages. Other biological mechanisms underlying the protective effect of Alc include the increase of paraoxonase activity, the reduction of lipoprotein (a) levels, the inhibition of blood clotting and platelet aggregation, the improvement of insulin sensitivity, the lowering of plasma homocysteine levels, the estrogen levels increase, and the reduction of stress. Moreover, moderate alcohol intake has been reported to confer protection against
inflammation, a process with a fundamental role in the initiation, progression, and the thrombotic complications of atherosclerosis. Such functions are extensively considered in other reviews (Agarwal, 2002; Imhof and Koenig, 2003).

**Side Effects of Alc**

The proposed cardiovascular benefits of Alc consumption must be evaluated against numerous adverse effects. Beyond two drinks per day, no further reduction in cardiovascular mortality has been observed (Gaziano et al., 2000). This appears to be due to increases in Alc-related medical disorders, such as 1) cardiovascular system—hypertension, cardiomyopathy, atrial and ventricular arrhythmias, and hyperlipidemia; 2) central nervous system—acute delirium, hepatic encephalopathy, cerebrovascular accidents, and cerebral atrophy; 3) muscle-skeletal system—myopathy and hyperuricemia; 4) gastrointestinal system—fatty liver, hepatitis, cirrhosis, hepatoma, and ascites; and 5) hematopoietic system—anaemia, coagulation abnormalities, leucopenia, and thrombocytopenia. Binge drinking is associated with impaired left ventricular function and arrhythmias (the most frequent paroxysmal atrial fibrillation—the “holiday heart syndrome”) (Greenspon and Schaal, 1983). An increased incidence in myocardial infarction on Mondays has been noted. Several explanations for this observation have been proposed, such as an increase in stress due to the transition from leisurely pace of life on weekends to a work schedule (Spielberg et al., 1996). Although this explanation may account for the higher occurrence of cardiovascular deaths on Mondays, another important factor is heavy or binge drinking during the weekend (Evans et al., 2000). Irregular heavy drinking is also related to stroke and sudden cardiac death (Rehm et al., 2003). These adverse effects of Alc can be attributed to increased clotting, rise in low density lipoprotein (LDL) cholesterol concentration, and lower threshold for ventricular fibrillation that occur with heavy drinking (Rehm et al., 2003).

Alc is one of the factors most frequently associated with increased liver enzyme activity in both animals (Kolovou et al., 2003, 2004, 2005) and humans (Kraemer et al., 2003). In our studies (Kolovou et al., 2003, 2004, 2005) with rats, Alc administration caused an increase in aspartate aminotransferase (AST) levels, similar to Kamimura et al. (1992) who observed 2- and 3-fold increases in plasma alanine aminotransferase and AST levels in Alc-fed male Wistar rats.

The association between Alc intake and Alc-induced liver disease in humans is well known. The Italian Dionysos study showed that Alc is suspected to cause the 23% of all cases of liver disease, with a dose-dependent increase in the risk of developing liver disease (Bellentani et al., 1997). Other investigators (Kraemer et al., 2003) also showed elevation of aminotransferase levels in humans. Conversely, investigators from Japan did not show a strong relationship between Alc consumption and serum enzyme activity, specifically AST and alanine aminotransferase (Nakamura et al., 1998).

The close relationship between ethanol consumption and liver function is due to the fact that more than 80% of ingested Alc is metabolized in the liver (Uzun et al., 2005). Alcohol is catabolized through two different pathways according to the mode of consumption (Fig. 2). After moderate consumption, ethanol is oxidized to acetaldehyde in the cytosol via the action of alcohol dehydrogenase by a concomitant conversion of NAD to reduced NADH. Acetaldehyde is further oxidized by aldehyde dehydrogenase to acetate (Lieber, 2003). During both the above oxidizing processes, nitric oxide radicals are also formed (Uzun et al., 2005). In the case of chronic heavy consumption, the microsomal ethanol-oxidizing system is activated. The enzyme P450 cyto-

![Fig. 2. The pathways of alcohol catabolism. ADH, alcohol dehydrogenase; MEOS, microsomal ethanol-oxidizing system; NO, nitric oxide; ROS, reactive oxygen species.](image-url)
Acetaldehyde is a highly reactive molecule that may alter the intracellular redox status generating oxidative stress (Dupont et al., 2000). Nitric oxide radicals and reactive oxygen species, also produced by ethanol breakdown, contribute to this latter condition (Lieber, 2003; Uzun et al., 2005). Oxidative stress leads to protein deactivation, inactivation of essential enzymes, damage of antioxidants, such as glutathione and vitamin E, triggering of inflammatory response, and alteration of fat breakdown (McCuskey et al., 1995; Lieber, 2003). Acetate released into the plasma is shown to decrease lipolysis in peripheral tissues by 53% and the whole-body lipid oxidation by 73% (Siler et al., 1999). Excess NADH concentration reduces fat breakdown and generates fatty acids giving rise to fat accumulation in liver, a fact that raises TG serum levels by increased production and secretion of VLDLs (Ginsberg et al., 1974). The chronic excessive consumption of Alc has been associated with hepatosteatosis, liver fibrosis, and cirrhosis. Liver cirrhosis is related with the overall volume of Alc intake (Kozarevic et al., 1983).

Furthermore, epidemiological data clearly show that increasing Alc drinking is associated with higher mean blood pressure and/or hypertension (Grobbee et al., 1999). On the other hand, the short-term lowering of blood pressure caused by Alc is also known. The effect of Alc on blood pressure varies according to chronicity and amount of intake. A J-shaped association has been proposed for the relationship between Alc and blood pressure (Gillman et al., 1995). Many studies support that there is a threshold of two to three drinks daily, below which Alc may exert a beneficial effect (Gillman et al., 1995). Many studies support that there is a threshold of two to three drinks daily, below which Alc may exert a beneficial effect (Gillman et al., 1995). Moreover, the metabolism of Alc may affect the breakdown and elimination of certain medications, including hypolipidemic drugs, through microsomal ethanol-oxidizing system and consequently contribute to harmful interactions (Dupont et al., 2000; Lieber, 2003).

As already stated, oxidation of Alc may provoke oxidative stress, alter peroxisome proliferators-activated receptor function (Huang et al., 1999), and induce potent inflammatory cytokines, such as tumor necrosis factor-α (McCuskey et al., 1995). In contrast, statins increase nitric oxide production and/or bioavailability, which may stabilize the hepatic microvascular inflammatory response to acute Alc ingestion, helping to protect the liver from ischemia and oxidative injury (Kolyada et al., 2001). In some cases, statins produce characteristic alterations in liver histopathology (e.g., peripoortal hepatocellular atypia) (Kolyada et al., 1999).

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P450 3A4 is only a minor contributor to overall ethanol metabolism (Bottorff and Hansten, 2000; Lieber, 2003).

Acute and chronic effects of ethanol are usually opposite in the context of drug metabolism. In contrast to chronic use, an acute dose of ethanol inhibits the metabolism of drugs, such as statins. Although this inhibition has been attributed, to some extent, to the competition for an at least partially shared microsomal detoxification pathway (Lieber, 1980), acute ethanol consumption mainly decreases drug metabolism indirectly by affecting the supply of NADPH (a cofactor necessary for the microsomal ethanol-oxidizing pathway) through excess NADH production in the ADH pathway (Fig. 2) (Thurman and Kauffman, 1979).

However, statin effectiveness was not affected in studies with concurrent chronic use of Alc. In contrast, Zdrenghea et al. (2004) observed that moderate Alc consumption, in combination with simvastatin, increased the beneficial effect upon HDL cholesterol after comparing 20 patients on simvastatin monotherapy (20 mg per day) with 20 patients on the same dose of simvastatin plus 30 g of Alc per day for a 2-week period. In addition, a study of the West of Scotland Coronary Prevention Study showed that subjects who drank >21 units of Alc per week seemed to have smaller reductions in LDL at all ages and a smaller increase in HDL cholesterol in response to statin treatment (Streja et al., 2002). Smit et al. (1995) found that Alc ingestion of 20 g per day for 6 weeks together with fluvastatin (40 mg per day) resulted in greater total and LDL cholesterol reduction. With regard to the safety of Alc and simvastatin chronic combination, in a study involving Wistar rats, no liver histopathological derangement was caused, whereas simvastatin decreased the Alc-induced TG and AST increased (Kolovou et al., 2003).

**Fibric Acid Derivatives**

Fibric acid derivatives (fibrates) lower plasma TG and raise HDL levels and thus are used to treat hypertriglyceridemia (Todd and Ward, 1988). Fibrates have a different mode of action than statins by reducing TG-rich lipoprotein precursors and favorably altering LDL and HDL composition (Scott, 1997). They also modulate thrombotic homeostasis in the blood (Capato, 1992). Fibrates bind to peroxisome proliferator-activated receptor-α, a nuclear receptor superfamily member, and induce β-oxidation of fatty acids in mitochondria (Tsutsumi and Takase, 2001).

Because of this latter effect, there are studies evaluating fibrates in the treatment of alcoholic fatty liver. Alc metabolism causes accumulation of fatty acids in the liver and a concomitant rise in serum TG levels (Ginsberg et al., 1974). Tsutsumi and Takase (2001) showed that hepatic TG content was significantly decreased in Alc-treated rats that were given fenofibrate. Hayashi (2000), on the other hand, suggested that simfibrate and clofibrate induced β-oxidation by peroxisome and increased H2O2 production, which led to augmented ethanol metabolism by catalase. This action of fibrates was also supported by Tsukamoto et al. (1996), who studied another fibrate, bezafibrate. However, fibrates can cause hepatotoxicity, hepatic peroxisome proliferation, and cancer in rats (Sausen et al., 1995). Nevertheless, in combination with Alc consumption, fibrates were shown to be safe for the liver after histopathological assessment in rats (Kolovou et al., 2004). With regard to humans, Tsutsumi and Takase (2001) observed a decrease in serum TG and total cholesterol levels after treatment with fenofibrate in alcoholic patients, although no fibrate-induced hepatotoxicity has been reported in humans (Sausen et al., 1995).

**Nicotinic Acid**

Nicotinic acid (NA) lowers plasma total cholesterol and TG levels, reducing VLDL and LDL cholesterol levels, and is also effective in raising HDL cholesterol (Tató et al., 1998). The plausible mechanism is that NA reduces the production of free fatty acids by inhibiting lipolysis in adipose tissue, which results in a reduced availability of substrate for VLDL synthesis in the liver (Tató et al., 1998). This speculation was confirmed by the identification of a G-protein-coupled receptor that is highly expressed in adipose tissue and to which NA is a high-affinity ligand (Wise et al., 2003). The binding of NA to its receptor activates a G-protein signal, which reduces cAMP concentrations and thus inhibits lipolysis. However, a “rebound” elevation in free fatty acids has been described previously (Karpe and Frayn 2004). Karpe and Frayn (2004) suggest that the effect of NA relies on the down-regulation of the activity of hormone-sensitive lipase. The lowering of free fatty acid concentration results in a reduction in triglyceride levels, which in turn leads to increased HDL cholesterol.

Another potential mechanism by which NA raises HDL cholesterol levels is through the stimulation of the ATP-binding cassette AI-mediated transfer of cholesterol (Rubic et al., 2004). It has also been suggested that NA directly inhibits the synthesis of apoB-containing lipoproteins in the liver (Tató et al., 1998).

Because NA reduces the production of free fatty acids, it could be speculated that its administration may also protect against ethanol-induced fatty liver. Sorrell et al. (1976) reported that NA not only does not protect against the ethanol-induced fatty liver but that it potentiates steatosis in rats. They attributed their findings to the fact that, after chronic ethanol administration, the source of the accumulated TG in liver is dietary fatty acids and not that from adipose tissue (Sorrell et al., 1976). Therefore, the inhibition of adipose tissue lipolysis by NA did not protect against ethanol-induced fatty liver. However, in a study of ours involving rats, Alc-induced secondary hypertriglyceridemia was alleviated after NA administration (Kolovou et al., 2005). Inhibition of hepatic steatosis and alcohol dehydrogenase by NA in ethanol-treated rats was also reported by Baker et al. (1973). It has been suggested that NA interacts with Alc (Bays and Dujo- vne, 1998), but it was also shown that, in experimental animals, it may reduce Alc-induced aspartate aminotransferase rises (Kolovou et al., 2005).

The administration of NA, however, can be accompanied by adverse effects in humans, which include flushing, itching, nausea, diarrhea, decreased glucose tolerance, hyperuricemia, and hyperhomocysteinemia (Meyler, 1998). Raised hepatic enzymes, cholestasis, and hepatocellular injury have also been reported after NA administration (Patterson et al., 1983). The tissue distribution of the NA receptor implies that the effects observed in the liver are secondary to alterations in fatty acid metabolism (Karpe and Frayn, 2004). The evidence suggests that, in humans, NA-induced toxicity is dose-related (Clementz and Holmes, 1987). However, NA, in the range of recommended human dose when coadministered...
with Alc in rats, did not disturb liver histopathology (Kolovou et al., 2005).

**Conclusions**

The beneficial or the detrimental effect of Alc on cardiovascular disease is explained by plausible physiological mechanisms. Light-to-moderate Alc consumption on a regular basis reduces cardiovascular risk without side effects; whereas irregular heavy drinking has a negative effect on all-cause mortality. It seems that all-cause mortality attributed to Alc is determined by both the pattern and the total volume of Alc intake (Rehm et al., 2003). An interesting aspect is that of population differences in Alc metabolic efficiency, which is likely to contribute to an individual’s susceptibility to Alc-related diseases. Studying genetic differences that potentially influence disease susceptibility among populations may provide insight into the mechanism(s) for the relationship between risk factor and disease, such as Alc and CHD (Hines, 2004).

Patients at high CHD risk could gain from the beneficial effects of Alc. Because most of these patients are on lipid-lowering drugs, a question of whether Alc drinking can be combined with hypolipidemic treatment arises. Attention should be paid to patients with alcoholic liver disease in which hepatic function is already disturbed; administration of lipid-lowering drugs may lead to aggravation of the existing disease. The prescription of hypolipidemic treatment to patients with chronic liver disease is under consideration, because little evidence exists concerning any further liver injury induced by such treatment. In cases where hypolipidemic therapy is required, the patients should be closely monitored. However, in patients with acute liver disease (acute viral hepatitis and alcoholic hepatitis), such drugs should not be administered until recovery (Russo and Jacobson, 2004).

More research is needed to determine whether the consumption of Alc in combination with hypolipidemic therapy should be recommended and if it is safe. Furthermore, there is a need to define the benefit and establish which drug behaves better in such a setting.

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Address correspondence to: Dr. Genovefa D. Kolovou, 1st Cardiology De-
partment, Onassis Cardiac Surgery Center, 356 Sygrou Ave., 176 74 Athens, Greece. E-mail: genkolovou@mail.gr

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