### CHAPTER 1- INTRODUCTION

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CHAPTER 1-INTRODUCTION

1.1 INTRODUCTION

Alcoholism represents one of the most serious worldwide socioeconomic and health problems. In speaking about the role of alcohol in society, Abraham Lincoln observed, "None seemed to think the injury arose from the use of a bad thing but from the abuse of a very good thing".

An alcoholic is a person who consumes an amount of alcohol capable of producing pathological changes (Criteria committee, 1972). The amount of alcohol capable of producing disease depends on a variety of factors, including genetic predisposition (Bailey et al., 1976), malnutrition (Mendenhall, 1984), and concomitant viral infections of the liver (Hall, 1985).

CAUSES OF ALCOHOLISM

This section reviews the causative factors involved in alcoholism as they operate on three levels: (1) Biochemical and psychological factors (2) Availability of alcohol and its attractiveness as determined by social attitudes and (3) Physiological changes as well as cultural attitudes. Alcohol provides a "pseudo-solution" to problems and anxieties. It is when this collapses, the alcoholic turns to religious solutions.

Marmot et al. (1981), Klatsky et al. (1992); and Renaud et al. (1999) reported association of heavy alcohol intake with a significant increase of all-cause and non-cardiovascular mortality rates especially by cirrhosis, cancer and violent deaths. They also reported that all-cause mortality rates are lower for moderate drinkers than for non-drinkers. This beneficial effect of moderate alcohol consumption might be explained by a rise of high density lipoprotein cholesterol (HDL-c) induced by alcohol consumption (Rimm et al., 1999), but also by other mechanisms such as alcohol anti-aggregation properties (Meade et al., 1985). The article by Gaziano et al. (1993) again raises the question whether, from the standpoint of one's health, moderate consumption of alcoholic beverages, usually defined as up to two standard drinks per day, is a good thing.

Heavy alcohol intake was reported to be associated with an increase in blood pressure by Milon et al. (1982) and Marmot et al. (1981)
Ethyl alcohol or ethanol, known commonly as alcohol, is the same whether the beverage is wine, beer, or hard liquor. Beverage alcohol is a drug that depresses the central nervous system, like barbiturates, sedatives, and anesthetics. Alcohol is not a stimulant. There is no question that the person who drinks alcohol seems stimulated. Speech becomes free and animated, social inhibitions may be forgotten, and the drinker can begin to act and feel more emotional. But these effects are misleading; the "stimulation" occurs only because alcohol affects those portions of the brain that control judgment. "Being stimulated" by alcohol actually amounts to a depression of self-control. A principal effect of alcohol is to slow down brain activity, and depending on what, how much, and how fast a person drinks, the result is slurred speech, hazy thinking, slowed reaction time, dulled hearing, impaired vision, weakened muscles and fogged memory. Alcohol is also classified as a food because it contains calories. The average drink has about the same calorie count as a large potato but, unlike a potato or any other food, alcohol has no nutritional value. The calories are empty.

1.2 USES OF ALCOHOL

Automotive:

Alcohol is often used as an automotive fuel (Owen and Coley, 1995). Ethanol and methanol can be made to burn more cleanly than gasoline or diesel. Alcohol was once commonly used as antifreeze in automobile radiators. To add to an internal combustion engine's performance, Methanol may be injected into turbocharged and supercharged engines. This cools the air intake charge, providing a denser air charge (Brinkman et al, 1994).

Scientific, medical, and industrial:

Alcohols are in wide use in industry and science as reagents or solvents. Because of its low toxicity and ability to dissolve non-polar substances, ethanol is often used as a solvent in medical drugs, perfumes, and vegetable essences such as vanilla. In organic synthesis, alcohols frequently serve as versatile intermediates.

Ethanol is often used as an antiseptic, to disinfect the skin before injections are given, often along with iodine. Ethanol-based soaps are now becoming common within restaurants and are particularly convenient as they do not require drying due to...
the volatility of the compound. Alcohol is also used as a preservative for specimens (Lodgson, 1994, Victor, 2005).

H1N1 virus and alcohol based hand rub:

Grayson et al., (2009), have reported that Hand Hygiene with alcohol-based hand rub or soap and water is highly effective in reducing influenza A virus on human hands, although Soap and water is the most effective intervention. Appropriate Hand Hygiene may be an important public health initiative to reduce pandemic and avian influenza transmission.

1.3 BIOCHEMISTRY

In chemistry, an alcohol is any organic compound in which a hydroxyl group (-OH) is bound to a carbon atom of an alkyl or substituted alkyl group. The general formula for a simple acyclic alcohol is C\textsubscript{n}H\textsubscript{2n+1}OH (Teschke and Lieber, 1978).

Generally, the word alcohol, when used alone, usually refers to ethanol, also known as grain alcohol or (older) spirits of wine. Ethanol is a very strong and unique smelling, colorless, volatile liquid formed by the fermentation of sugars. It also often refers to any beverage that contains ethanol (see alcoholic beverage). It is the most widely used depressant in the world, and has been for thousands of years. This sense underlies the term alcoholism (addiction to alcohol).

1.4 PHYSIOLOGY

Basics of alcohol metabolism:

Alcohol is not digested like other foods. Instead of being converted and transported to cells and tissues, it avoids the normal digestive process and goes directly to the bloodstream. About 20 percent of the alcohol is absorbed directly into the blood through the stomach walls and 80 percent is absorbed into the bloodstream through the small intestine (Paul et al., 2007).

Ethanol is extensively metabolized in the liver, leading to the generation of acetaldehyde by the enzymatic activity in cytosol, microsomes and peroxisomes. Acetaldehyde is further oxidized to acetate by acetaldehyde dehydrogenase in the mitochondria, which results in the generation of free radicals/reactive oxygen species (ROS) (Mayes, 1993, Charness, 1993; Zima et al., 2001, Husain et al., 2005).
Oxidation of ethanol by alcohol dehydrogenase generates NADH and NADH-dependent production of ROS by various organelles increases after chronic alcohol consumption. These ROS can cause cellular damage until they are removed by the antioxidant system (Soman and Husam, 1997, Reddy et al., 1999).

Alcohol dilutes itself in the water volume of the body in order to travel through the system. Those vital organs, like the brain, that contain a lot of water and need an ample blood supply are particularly vulnerable to the effects of alcohol. Alcohol's dilution in the body does cut its effect somewhat. There one important biological difference between men and women comes into play: Muscle tissue contains more water than fat tissue, so men—who have more muscle and less fat on the average than women—can have about 10 percent more water in their bodies. If a lean man and a lean woman of equal weight consume the same amount of liquor, the woman is more adversely affected for this and other reasons.

The brain, liver, heart, pancreas, lungs, kidneys, and every other organ and tissue system are infiltrated by alcohol within minutes after it passes into the bloodstream. The strength of the drink will have a significant effect on absorption rates, with higher concentrations of alcohol resulting in more rapid absorption. Pure alcohol is generally absorbed faster than diluted alcohols, which are, in turn, absorbed faster than wine or beer.

Alcohol taken in concentrated amounts can irritate the stomach lining to the extent that it produces a sticky mucous which delays absorption. The pylorus valve which connects the stomach and small intestine may go into spasm in the presence of concentrated alcohol, trapping the alcohol in the stomach instead of passing it on to the small intestine where it would be more rapidly absorbed into the bloodstream. The drinker who downs several straight shots in an effort to get a quick high may actually experience a delayed effect. Finally, the temperature of the beverage affects its absorption, with warm alcohol being absorbed more rapidly than cold alcohol.

**Measurement of effect by blood alcohol level (BAL):**

The drinker's blood alcohol level rises as a factor of the relationship among the amount of alcohol consumed, body size and proportion of body fat, the amount of food in the stomach, and what is mixed with the alcohol. The BAL rises more rapidly in those who drink on an empty stomach. Water and fruit juices slow the absorption process, while carbon dioxide speeds it up. The carbon dioxide in champagne and
carbonated mixers such as Cola, and soda water rushes through the stomach and intestinal walls into the blood stream, carrying alcohol with it and creating a rapid rise in BAL. A 0.08 BAL, for example, indicates approximately 8 parts alcohol to 10,000 parts other blood components. When a person drinks more alcohol than his or her body can eliminate, alcohol accumulates in the blood stream and the BAL rises.

Elimination of alcohol from a healthy adult body occurs at an average rate of approximately ½ to ¾ ounce per hour, the equivalent of 1 ounce of 100-proof whiskey, one large beer, or about 3 to 4 ounces of wine. When blood alcohol concentrations reach very high levels, the brain’s control over the respiratory system may be paralyzed A 0.30 BAL is the minimum level at which death can occur; at 0.40 the drinker may lapse into a coma At 0.50 BAL, respiratory functions and heartbeat slow drastically, and at 0.60 most drinkers are dead.

1.5 BODY SYSTEMS AND EFFECTS

Acute or chronic alcohol consumption causes degeneration in different internal organs and systems of adults (Mezey, 1985; Persson et al., 1990; Watabiki et al., 2000; Benicky et al., 2000; Fortunato and Gates, 2000).

Alcohol has been widely consumed through the ages because of its perceived benefits as a social lubricant and for relaxation, mood alteration, and sensory pleasure. But long-term consumption of large amounts is harmful, leading to addiction and fatal or nonfatal injuries. It can cause cirrhosis of the liver; pancreatitis, gastritis; hypertension; cardiomyopathy, dysrhythmia; hemorrhagic stroke; degenerative nervous system conditions; cancers of the mouth, pharynx, larynx, esophagus, and liver; and fetal damage.

Excessive alcohol intake may cause serious harm to others, as is exemplified by the all too common problem of drunk driving. Concern about these dangers often leads to emotional denials that alcohol might have any benefits, particularly by those who have experienced or seen its bad effects (Yano et al., 1977, Stampfer et al., 1988; Boffetta and Garfinkel, 1990, Klatsky et al., 1990).

The Liver:

Located in the upper-right side of the abdomen, the liver is the body’s largest glandular organ. Its complex functions are associated with dozens of processes of body chemistry and metabolism. It produces the bile that helps digest fatty foods; it
manufactures heparin, an anticoagulant, it stores and releases sugar. The liver also produces antibodies that help ward off disease, and it cleanses the body of poisons, including alcohol. With small amounts of alcohol, this cleansing can happen effectively. When the amount of alcohol is high, imbalances are created which can lead to hypoglycemia (low blood sugar), hyperuricemia (as in arthritis or gout), fatty liver (which may lead to hepatitis or cirrhosis), and hyperlipemia (build-up of fats sent to the bloodstream; which leads to heart problems) (Ajmo et al., 2008).

The Central Nervous System:

The central nervous system (CNS) includes the brain, the spinal cord, and the nerves originating from it. Sensory impulses are transmitted to the CNS and motor impulses pass from it. When alcohol acts on the CNS, intoxication occurs, affecting emotional and sensory function, judgment, memory and learning ability. Smell and taste are dulled. The ability to withstand pain increases as the BAL rises.

Different parts of the brain seem to be affected by alcohol at different rates, creating alternate periods of restlessness and stupor. Long-term effects of alcohol on the central nervous system include tolerance, dependency, and irreversible damage. Changes in tolerance for alcohol, and the alcoholic drinker's dependency on alcohol, demonstrate that changes occur in the brain.

With each drinking episode, central nervous system functions deteriorate in a predictable sequence, beginning with intellectual functioning, followed by disturbances in sensory and motor control. Last affected are the automatic biological functions, such as breathing and heart action (Nixon and Crews, 2002; Herrera, 2003; Crews, 2003).

The brain is the organ that is most affected by alcohol, and proves that it is being damaged through the drinker's behavior changes and emotional distress. Three noticeable effects of alcohol injury to the brain memory loss, confusion, and augmentation (Augmentation is a physiological response to alcohol which results in hyper-alertness to normal situations, perceiving light as brighter or sounds as louder than usual, or the drinker's becoming extremely sad or angry for no apparent reason.) The drinker's rapid mood swings and emotional and behavioral instability can be brought under control by stopping drinking.

Blackouts, or loss of memory for a period during drinking, are a physical effect of alcohol on the brain (Schottenbauer et al., 2007) They occur as alcohol cuts
off the supply of oxygen to the brain. Lack of oxygen supply to the brain can kill tens of thousands of brain cells every time a person becomes intoxicated.

Another effect of alcohol on the brain is the "learned behavior syndrome"; when a behavior is learned under the influence of alcohol, the drinker sometimes must re-learn that behavior after stopping drinking.

The Blood:

One effect of drinking alcohol is "blood-sludging" where the red blood cells clump together causing the small blood vessels to plug up, starve the tissues of oxygen, and cause cell death. This cell death is most serious, and often unrecognized, in the brain. With this increased pressure, capillaries break; create red eyes in the morning, or the red, blotchy skin seen on the heavy drinker's face. Blood vessels can also break in the stomach and esophagus leading to hemorrhage, even death.

Other effects of alcohol on the blood include: anemia, sedation of the bone marrow (which reduces the red and white blood count, and weakens the bone structure); lowered resistance to infection; and a decrease in the ability to fight off infections.

The Gastrointestinal Tract:

Mezey (1985) stated that high alcohol intake is a known cause of diarrhea, other gastrointestinal symptoms and in advanced states, decreases in body weight. Generally, it is accepted that, in individuals with high alcohol consumption, malnutrition develops depending on the possible changes in intestinal absorption mechanisms and dysfunction of some organs such as the liver and the pancreas (Persson et al., 1990).

The stomach, the small and large intestines, and the pancreas are each affected by alcohol. Alcohol increases acid in the stomach. That can result in gastritis or stomach or intestinal ulcers. The pancreas produces insulin which is necessary to regulate the amount of sugar in the blood. Drinking causes a steep rise in the blood sugar; the pancreas responds by producing insulin which causes a fast drop in blood sugar and the symptom of low blood sugar or hypoglycemia. 70-90% of alcoholics suffer to some degree from the disorder of hypoglycemia, chronic low blood sugar, as a long term effect of their drinking. Symptoms of hypoglycemia can include dizziness, headaches, lack of ability to concentrate, depression, anxiety, light-
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headedness, tremors, cold sweats, heart palpitations, loss of coordination, and upset stomach. In time, the drinker's overworked pancreas may stop producing insulin and diabetes can result. Conversely, a person with a family history of diabetes may be more vulnerable to problems with alcohol.

The Muscles:

Alcohol reduces blood flow to the muscles, including the heart, causing muscle weakness and deterioration. One outcome is cardiomyopathy (sluggish heart) which is common in alcoholics. Another outcome, arrhythmia (irregular heartbeat), or "holiday heart" is often treated in emergency wards after several days of party drinking. Muscle aches are a common symptom of excessive-drinking "hangovers" (Femandez-Sola et al., 1995).

The Endocrine System:

This system controls the body's hormones and includes the pineal, pituitary, thyroid, and adrenal glands, and the ovaries or testes. Alcohol sedates these glands, resulting in under-production of hormones, effects include increased susceptibility to allergies. Alcohol can affect sexual functioning in various ways. In low doses, it lowers inhibitions and may make a person feel sexier; but in higher doses, it can decrease sexual functioning in men, by decreasing the frequency of erections, decreasing the maintenance of erections, decreasing penile size during erection, and increasing the amount of time between erections, in women by interfering with normal processes of sexual stimulation, and blocking orgasmic response. With chronic and prolonged use of alcohol in men, there is shrinkage of sex glands and an increase of the "female hormone" estrogen. This produces secondary sexual characteristics, such as enlarged breasts and a decrease in body hair. Prolonged use of alcohol can cause infertility in both men and women. In men, reproductive hormones are responsible for sexual maturation, sperm development and thus fertility, and various aspects of male sexual behavior. In women, hormones promote the development of secondary sexual characteristics, such as breast development and distribution of body hair, regulate the menstrual cycle; and are necessary to maintain pregnancy. Chronic heavy drinking can interfere with all these functions. It's most severe consequences in both men and women include inadequate functioning of the
testes and ovaries, resulting in hormonal deficiencies, sexual dysfunction, and infertility (Emanuele et al., 1993, Mello et al., 1993).

Alcohol is directly toxic to the testes, causing reduced testosterone levels in men. In a study of normal healthy men who received alcohol for 4 weeks, testosterone levels declined after only 5 days and continued to fall throughout the study period (Gordon et al., 1976). Prolonged testosterone deficiency may contribute to a "feminization" of male sexual characteristics, for example breast enlargement (Bannister and Lowosky, 1987).

Alcohol can interfere with calcium and bone metabolism in several ways. Acute alcohol consumption can lead to a transient PTH deficiency and increased urinary calcium excretion, resulting in loss of calcium from the body (Laitinen et al., 1991). Chronic heavy drinking can disturb vitamin D metabolism, resulting in inadequate absorption of dietary calcium (Bjorneboe et al., 1988).

Cytotoxic effect of alcohol on skin:

Because of the cytotoxicity of alcohol, researchers have suggested that one should use it topically at a dilution of up to 1:1,000 of commonly used concentration and for a short period only (Pyo et al., 2003).

Alcohol used as an antiseptic or preservative can effectively kill bacterial and fungal cells, but will just as effectively kill healthy cells. Compromised skin barrier function increases susceptibility for damage of the skin and permeability by other chemicals. (Smith and Maibach, 1995) Substances applied to the skin can diffuse across the protein/lipid barrier into keratinocytes below. Facial skin is even more permeable, which is of consequence because many alcohol-containing cosmetics, creams, lotions, and gels are commonly regularly applied to the face (Neuman et al., 2002). Ethanol can be a skin allergen in immediate and delayed hypersensitivity by external or internal exposure and can produce subjective irritation, irritant contact dermatitis, and non-immunologic contact urticaria (Ophaswongse and Maibach, 1994).

Electron microscopy revealed that alcohol exposed human skin cells, even at very low concentrations caused organelle damage, condensed chromatin, decreased cell size and increased apoptotic bodies (cellular suicide) (Neuman et al., 2002).

Psoriasis and eczema are associated with excessive alcohol exposure (Higgins and, du Vivier, 1992). Canadian collaborators of mine investigated whether alcohol plays a role in the pathogenesis of psoriasis by up-regulating humoral pro-
inflammatory cytokines and concluded that in normal human skin cells, toxicity and psoriasis-causing inflammatory responses are enhanced by a concentration as low as 40 mM alcohol (Shear N, 1999). Furthermore, paradoxically, alcohol concentrations below levels that induce cytotoxicity (0.1–0.5%), may be immuno-suppressive by inhibiting the inflammatory response and thereby impairing associated cellular immune responses to infectious challenge, thereby increasing the risk of infection for several hours (Saeed et al., 2004).

That alcohol exposure is toxic to human skin cells, is clearly established. The skin, like the liver, is furthermore one of the few organs capable of metabolising alcohol to another cytotoxic chemical, acetaldehyde. Glutathione was greatly reduced in cells consecutively exposed to the alcohol (a mere one day after another, compared to perpetually, as occurs in real life use of skin care products) Glutathione is important for protection from oxidative damage. Lower levels increase vulnerability of skin cells to oxidative stress by free radicals and other damaging reactive oxygen species. Conclusion: Alcohol is toxic to human skin cells. Repeated exposure from skin products may threaten cell viability. The cytotoxic risks associated with prolonged exposure to alcohol deserve investigation. Alcohol-free personal care products may prove less harmful to the skin.

There are several mechanisms by which alcohol is cytotoxic. Some involve direct cytotoxic action of alcohol itself, without being metabolised, primarily via the generation of oxygen radical intermediates and free radicals (Mufti et al., 1996). The most toxic action of alcohol results from its even more toxic metabolite, acetaldehyde (approximately 30 times more toxic), which is produced from alcohol by several enzymes in human skin (Robert Dudley and Michael Dickinson, 2004). Somewhat reduced toxicity might result from further breakdown of acetaldehyde (which is related to formaldehyde) to acidic toxic oxidation transition products, including carboxylic acids. If acetaldehyde is not eventually efficiently converted into acetic acid (the acid in vinegar), severe toxicity can result (Cheung et al., 2003).

Researchers have detected important classes of enzymes involved in the biotransformation of both alcohols and aldehydes in human skin (Cheng et al., 1999) that present skin sensitization hazards with potency relative to the extent of metabolism in the skin. Alcohol dehydrogenase (ADH) enzymes catalyze the interconversion of alcohols and aldehydes and convert aldehydes to acids. Aldehyde dehydrogenase (ALDH) enzymes catalyze the oxidation of aldehydes to carboxylic
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Acids. The enzymes are present in human skin, predominantly in the epidermis and appendages (sebaceous glands and hair follicles). These enzymes in human skin have toxicological significance with respect to the metabolism of xenobiotic (topically applied solvent tincture and preservative) ethyl alcohol and the resultant aldehydes.

The enzymatic breakdown of alcohol results in the generation of the toxic reactive molecule, acetaldehyde and as a byproduct, highly reactive oxygen radicals that interact with lipid molecules in cell membranes and via lipid peroxidation, generate additional reactive molecules, especially malondialdehyde and 4-hydroxy-2-nonenal. These interact with proteins, lipids and DNA to form adducts (hybrids) that impede the normal functions of proteins and induce harmful immune responses. These effects can lead to cellular dysfunction, cell damage and cell death (Tuma et al., 2004).

Collagen, the major protein in connective tissue (including the skin and loss of which results directly in wrinkles), is one protein preferentially damaged by alcohol aldehydes. Cross-linking is a process by which “molecular bridges” are formed between “reactive sites” on different molecules. Acetaldehyde induced cross-links tie up affected molecules and interfere with their normal vital functions, which may even be completely blocked. This process of cross-linking is largely responsible for the visible age-related changes in human skin that make it inflexible, sagging, and wrinkled and dry (Fowkes, 2004).

Alcohol exposure is highly conducive to the generation of oxygen free radicals and the subsequent attack of fragile polyunsaturated lipids, thereby producing cytotoxic lipid peroxidation products. Aberrations in phospholipid and fatty acid metabolism, changes in cellular redox state, disruptions of the energy state, and increased production of reactive oxygen metabolites are implicated in cellular damage resulting from both acute (occasional) and chronic (ongoing) exposure to alcohol. Non-oxidative metabolism of alcohol is furthermore an additional toxic mechanism by which alcohol affects membrane structure and compromises cell function (Baker and Kramer, 2004).

Alcohol exposure can be directly involved in the production of reactive oxygen species (ROS) and reactive nitrogen species (RNS), which form an environment favourable to oxidative stress. ROS and RNS play an important role in alcohol cytotoxicity via DNA damage, lipid peroxidation and protein modification. Alcohol-induced oxidative stress is linked to the metabolism of ethanol involving
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both microsomal and mitochondrial systems. Alcohol exposure results in the depletion of GSH levels and decreases antioxidant activity. It elevates malondialdehyde, hydroxyethyl radical and hydroxynonenal protein adducts. These cause the modification of all biological structures and consequently result in serious malfunction of cells and tissues (Das and Vasudevan, 2007).

Carcinogenic potentials of alcohol:

Alcohol can generate oxygen radical intermediates and free radicals, leading to lipid peroxidation, which promotes tumours (Mufti et al., 1993). Observations from experimental carcinogenesis and clinical studies indicate that though it cannot initiate carcinogenesis by itself, alcohol acts as a tumour promoter. Alcohol is cytotoxic and the injury induced may cause cell atrophy or cell death and sometimes, subsequent cell proliferation, culminating in some facet of neoplastic development. Studies suggest that long-term alcohol exposure favours malignant development of chemically induced lesions. Thus, alcohol has can play an active role where carcinogenesis stimulus are further developed by a dysplastic proliferative response and possibly an increase in malignancy (Ronald Watson, 1992).

Ethyl alcohol is a very potent radiomimetic agent, producing chromosome aberrations comparable to those induced by ionizing radiation. A concentration of 0.12 per cent alcohol produces a small increase over controls, and 0.25 per cent induces a substantial increase. A concentration of only 0.5 per cent alcohol was equivalent to about 20 rad/day of chronic gamma radiation, or an accumulated dose of 75 rad. To put this in perspective, the Atomic Energy Commission maximum permissible dose for radiation workers is a mere 0.1 rad per week, and for the general public, a minuscule 0.01 rad per week (Sax and Sax, 1966). DNA reactions with alcohol occur under physiological conditions in the presence of activating agents such as free radicals and exposure to UV or visible light (Fraenkel-Conrat and Singer, 1988).

The European Chemicals Bureau has proposed the classification of alcohol as a mutagen (ECBI/74/95-Add 3) under the Dangerous Substances Directive (67/548/EEC) and the German Commission for the Investigation of Health Hazards of Chemical Compounds classified it as a Category 2 Mutagen. The genetic effects are mostly due to the metabolite acetaldehyde, produced in the liver and skin (Philips and Jenkinson, 2001). Alcohol has been demonstrated to be carcinogenic for various
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organs and tissues and must be considered a multipotential carcinogenic agent (Soffritti et al., 2002).

Alcohol itself is not a carcinogen but under certain conditions is a co-carcinogen and/or tumour promoter. Alcohol exposure inhibits natural killer (NK) cell activity and reduces NK cell number. A major impact of alcohol on the immune system favoring tumour development is undisputed (Poschl and Seitz, 2004). Alcohol can also induce the cytochrome P450 enzyme that works with alcohol dehydrogenase to oxidise alcohol. This enzyme also forms dangerous reactive oxygen species that can activate environmental pro-carcinogens into carcinogenic forms (Goodsell, 2006).

Alcohol may also stimulate carcinogenesis by inhibiting DNA methylation (Setz, 2007). Experimental alcohol exposure results in increases in VEGF mRNA and its receptor protein levels in mammalian melanoma, with significantly increased melanoma growth (Tan et al., 2007).

Acetaldehyde is produced by the oxidation of ethyl alcohol in the body, including in the skin of humans. Mere micromolar concentrations of acetaldehyde, causes a wide range of cytopathic effects associated with multistep carcinogenesis. Acetaldehyde cytotoxicity causes comparatively higher genotoxicity and inhibits DNA repair more readily than other major aldehydes in tobacco smoke, automotive emissions and the manufacturing of plastics (Grafstrom et al., 1994). According to the International Agency for Research on Cancer there is sufficient evidence for acetaldehyde as a carcinogen in experimental animals (Lyons, Acetaldehyde. Monograph, 1995). Acetaldehyde is genotoxic, inducing gene mutations, clastogenic effects, and sister-chromatid exchanges in mammalian cells in the absence of exogenous metabolic activation. There is indirect evidence from in vitro and in vivo studies to suggest that it can induce protein-DNA and DNA-DNA cross-links (Rieger and Michaelis, 1960; Cortes et al., 1986).

The metabolism of alcohol leads to the generation of acetaldehyde and free radicals. Acetaldehyde is carcinogenic and mutagenic, binds to DNA and proteins, results in hyper-proliferation and is predominantly responsible for alcohol-associated carcinogenesis. It causes mutations and gross chromosomal aberrations and interferes with the DNA repair machinery. Acetaldehyde also binds rapidly to cellular proteins and DNA, resulting in morphological and functional cellular impairment. Binding to DNA triggers replication errors and/or mutations. These acetaldehyde-associated effects occur at concentrations as low as 40 μmol/l, which are similar to...
Acetaldehyde is however even more toxic than alcohol, but may, under favourable circumstances, be more safely oxidized to acetate by aldehyde dehydrogenase. However, alcohol exposure carcinogenesis involves these same enzymes, the acetaldehyde formed in the first step being the major culprit, constituting a reactive compound that forms covalent complexes with proteins and DNA and thus may act as a mutagen. Alcohol can also induce the cytochrome P450 enzyme that works with alcohol dehydrogenase to oxidize alcohol. This enzyme also forms dangerous reactive oxygen species that can activate environmental pro-carcinogens into carcinogenic forms (Goodsell, 2006). Acetaldehyde is one of 13 carcinogens accounting for approximately 23% of the carcinogenic effects of tobacco smoking (Sanner and Dybärg, 2007)

1.6 TERMS TO UNDERSTAND
Heavy Drinker and Chronic Drinker:

Many terms are applied to a drinker's relationship with alcohol. Use, misuse, heavy use, abuse, addiction, and dependence are all common labels used to describe drinking habits, but the actual meaning of these words can vary greatly depending upon the context in which they are used. Even within the medical field, the definition can vary between areas of specialization The introduction of politics and religion further muddles the issue

Use refers to simple use of a substance. An individual who drinks any alcoholic beverage is using alcohol. Misuse, problem use, and heavy use do not have standard definitions, but suggest consumption of alcohol to the point where it causes physical, social, or moral harm to the drinker. The definitions of social and moral harm are highly subjective and therefore differ from individual to individual

Within politics, abuse is often used to refer to the illegal use of any substance. Within the broad field of medicine, abuse sometimes refers to use of prescription medications in excess of the prescribed dosage, sometimes refers to use of a prescription drug without a prescription, and sometimes refers to use that results in long-term health problems. Within religion, abuse can refer to any use of a poorly regarded substance. The term is often avoided because it can cause confusion with audiences that do not necessarily share a single definition.
Chronic alcoholism is a pathologic condition resulting from the habitual use of alcohol in excessive amounts. The syndrome involves complex cultural, psychological, social, and physiologic factors and usually impairs an individual's health and ability to function normally in society. Symptoms of the disease include anorexia, diarrhea, weight loss, neurologic and psychiatric disturbances (most notably depression), and fatty deterioration of the liver, sometimes leading to cirrhosis. Treatment depends on the severity of the disease and its resulting complications; nutritional therapy, use of tranquilizers in the detoxification process, and hospitalization may be necessary.

Remission is often used to refer to a state where an alcoholic is no longer showing symptoms of alcoholism. The American Psychiatric Association considers remission to be a condition where the physical and mental symptoms of alcoholism are no longer evident, regardless of whether or not the person is still drinking. They further subdivide those in remission into early or sustained, and partial or full. Some groups do not recognize remission. Instead, these groups use the term recovery to describe those who have completely stopped consumption of alcohol and are addressing underlying emotional and social factors.

Tolerance. As people drink, their tolerance for alcohol may increase. They might seem to be able to "handle" alcohol better and need more to achieve the same effect as before. The liver does not become more tolerant, and is damaged over the course of time, leading to poor liver function and a noticeable decrease in tolerance, or "reverse-tolerance". A heavy drinker's reverse-tolerance is a sign of late-stage alcoholism.

Withdrawal. The effects of alcohol on the body account for the sick, uncomfortable, shaky feelings following a period of drinking. Withdrawal symptoms vary in intensity according to the amount and prolonged frequency of drinking. Symptoms of alcohol withdrawal include:

- Hang over's -- fairly common result of overindulging-- headache, fatigue, thirst, and nervousness. There may be nausea and abdominal cramping. Diagnosed alcoholics report fewer hangovers than drinkers who are non-alcoholic, this may be because they have learned to ignore the symptoms.
- Sleep disturbance -- waking up earlier than usual after expecting to "sleep it off," being unable to fall asleep, disturbed dreaming.
❖ Irritability, anxiety, and restlessness -- all caused by the irritant effects of alcohol
❖ Tremors, or "morning shakes"-- Tremors will clear after several days of abstinence, if there is no permanent damage to the nervous system.
❖ physical weakness, rapid heart rate,
❖ mental sluggishness
❖ difficulty thinking clearly or flexibly
❖ All the above are lingering evidence of alcohol's impact on muscles, heart and brain. For the drinker with only a mild degree of physical dependence, withdrawal effects may not extend beyond the symptoms listed above.

Some drinkers experience second stage withdrawal, marked by:
❖ Convulsions -- seizures usually occur between 12 and 48 hours of the last drink. There may be a loss of consciousness and body control.

Third stage withdrawal symptoms involve:
❖ Alcoholic hallucinosis and delirium tremens -- auditory, visual and tactile hallucinations occur. This period may last for three to four days, during which the de-toxifying person is in a severe state of agitation, is often completely disoriented and sleeps little, if at all. The delusions are almost always terrifying and may produce violent behavior. There is a 10%-20% mortality rate associated with this stage of withdrawal. Detoxification of the acutely ill alcoholic requires medical supervision.

Alcohols often have an odor described as 'biting' that 'hangs' in the nasal passages. Ethanol in the form of alcoholic beverages has been consumed by humans since pre-historic times, for a variety of hygienic, dietary, medicinal, religious, and recreational reasons. While infrequent consumption of ethanol in small quantities may be harmless or even beneficial, larger doses result in drunkenness or intoxication (which may lead to a hangover as the effect wears off) and, depending on the dose and regularity of use, can cause acute respiratory failure or death and with chronic use has medical repercussions. Because alcohol impairs judgment, it can often be a catalyst for reckless or irresponsible behavior.

Alcohol withdrawal is characterized by neuropsychiatric excitability and autonomic disturbances similar to other sedative-hypnotic drugs. Dependence on
other sedative-hypnotics increases the severity of the withdrawal syndrome (Sanna et al., 2003).

The severity of the alcohol withdrawal syndrome can vary from mild symptoms such as mild sleep disturbances and mild anxiety to very severe and life threatening including delirium, particularly visual hallucinations in severe cases and convulsions (which may result in death) (Liskow et al., 1989) The severity of alcohol withdrawal depends on various factors including age, genetics and most importantly degree of alcohol intake and length of time the individual has been misusing alcohol for and number of previous detoxifications (Howard, 1988) The acute phase of the alcohol withdrawal syndrome can also occasionally be protracted. Protracted delirium tremens has been reported in the medical literature as a possible but unusual feature of alcohol withdrawal (Miller, 1994).

Most patients undergoing alcohol withdrawal can be treated safely and effectively as outpatients Pharmacologic treatment involves the use of medications that are cross-tolerant with alcohol. Benzodiazepines, the agents of choice, may be administered on a fixed or symptom-triggered schedule. Carbamazepine is an appropriate alternative to a benzodiazepine in the outpatient treatment of patients with mild to moderate alcohol withdrawal symptoms Medications such as haloperidol, beta blockers, clonidine, and phenytoin may be used as adjuncts to a benzodiazepine in the treatment of complications of withdrawal. Treatment of alcohol withdrawal should be followed by treatment for alcohol dependence (Max et al., 2004).

1.7 SPECIAL CONCERNS OF WOMEN

Female drinkers reach higher blood alcohol levels (BAL's) faster because of less water and fatter in the body and because of differences in digestive enzymes. Women develop alcohol-related disorders such as brain damage, cirrhosis and cancers at lower levels of drinking than men It is also known that the menstrual cycle affects alcohol metabolism in women. Women have been shown to develop their highest BAL's immediately before menstruating and their lowest on the first day of menstruation. This can be related to hormone level shifts. There is evidence which shows that premenstrual syndrome with its emotional and physical discomfort and destabilized blood-sugar levels can trigger excessive drinking by some women.
Liver Damage: Compared with men, women develop alcohol-induced liver disease over a shorter period of time and after consuming less alcohol (Tuyns, 1984, Gavaler, 1995). In addition, women are more likely than men to develop alcoholic hepatitis and to die from cirrhosis (Hall, 1995). Animal research suggests that women’s increased risk for liver damage may be linked to physiological effects of the female reproductive hormone estrogen (Ikejima, 1998).

Brain Damage: Views of the brain obtained by magnetic resonance imaging (MRI) suggest that women may be more vulnerable than men to alcohol-induced brain damage. Using MRI, researchers found that a brain region involved in coordinating multiple brain functions was significantly smaller among alcoholic women compared with both nonalcoholic women and alcoholic men. These differences remained significant after measurements were adjusted for head size (Hommer, 1996). Conversely, a study measuring metabolic energy utilization in selected brain regions found a significant difference between alcoholic and nonalcoholic men but no significant difference between alcoholic and nonalcoholic women (Wnag, 1998). These results are not consistent with a greater vulnerability to alcoholic brain damage in women. However, the female alcoholics reported less severe alcohol use compared with the male alcoholics studied (Wang, 1998).

Heart Disease: Men and women who consume one or two alcoholic drinks per day have a lower death rate from coronary heart disease (e.g., heart attacks) than do heavier drinkers and abstainers, as discussed in Alcohol Alert No. 45, “Alcohol and Coronary Heart Disease” (Rockville, 1999). Among heavier drinkers, research shows similar rates of alcohol-associated heart muscle disease (i.e., cardiomyopathy) for both men and women, despite women’s 60 percent lower lifetime alcohol use (Urbano-Marquez, 1995).

Breast Cancer: Many studies report that moderate to heavy alcohol consumption increases the risk for breast cancer (Smith-Warner, 1998), although one recent study found no increased breast cancer risk associated with consumption of up to one drink per day, the maximum drinking level reported by most women (Zhang, 1999).
FETAL ALCOHOL SYNDROME (FAS) and FETAL ALCOHOL EFFECT (FAE)

Oyama et al. (2000) and Butters et al. (2000) reported the effect of maternal alcohol consumption on different organs and systems of the developing fetus. Important functional disorders of these organs and systems occur frequently because of these negative effects.

Furthermore, maternal alcohol during gestation is known to cause fetal growth retardation in humans and laboratory animals (Lin, 1981), an effect persisting for a long period after parturition (Sanchis-Segura et al., 2000; Juarez et al., 2000).

Children of alcoholics (COAs) also suffer from various deleterious effects. Alcoholism affects the entire family. Living with a non-recovering alcoholic in the family can contribute to stress for all members of the family. Each member may be affected differently. Not all alcoholic families experience or react to this stress in the same way. Children raised in alcoholic families have different life experiences than children raised in non-alcoholic families. Many children of alcoholics experience other family members as distant and non-communicative. Children of alcoholics may be hampered by their inability to grow in developmentally healthy ways. Children of alcoholics are four times more likely than non-COAs to develop alcoholism.

Women who drink during pregnancy risk the development of both mental and physical defects in their children. Effects on the child can include: growth deficiencies, poorly formed bones and organs, heart abnormalities, cleft palate, retarded intellect, delayed motor development, poor coordination, behavior problems, and learning disabilities. Smoking cigarettes, combined with alcohol use, will increase the chance of birth defects. Use of alcohol increases the chance of miscarriage. It is best that a woman avoid alcohol, cigarettes, caffeine, and other drugs entirely during pregnancy. Antabuse is not a suitable treatment for the pregnant or potentially pregnant alcoholic woman; it interferes with maternal liver function and may cause harm to the developing fetus.

Since harm to the infant may result even before a woman realizes that she is pregnant, women who might become pregnant need to be particularly cautious about what they consume.
1.8 NUTRITIONAL OVERVIEW

Secondary Diabetes:

Diabetes can result from prolonged, excessive use of alcohol. Because it is caused by drinking and not from a genetic disorder, it is called "secondary" diabetes. The symptoms are identical to genetic or "primary" diabetes. Abstinence from alcohol is a vital part of treatment for this disorder.

Alcohol and diabetes:

It is possible for alcohol to cause diabetes (though most cases of diabetes are not caused by alcohol). Alcohol abuse is the cause of 80% of cases of chronic pancreatitis and around 1 in 3 people with chronic pancreatitis develop diabetes.

Type 2 can be due to over weight or unfit but also normal weight, normally adults, can get type 2. They are different conditions but are treated similarly. Type 1 sufferers automatically need insulin injections or via a pump, and type 2 only sometimes need extra insulin. Other times it is dealt with by diet and/or tablets. Type 2 is even more strongly linked to genetics and family history than type 1 diabetes. A family history of type 2 diabetes and obesity are the main risk factors in type 2 diabetes.

DM in alcoholic pancreatitis:

The most direct mechanism by which alcohol can cause diabetes is through creatic destruction (Shimizu, 2008). Most cases of acute alcoholic pancreatitis seem to be associated with the development of chronic alcoholic pancreatitis (Skinazi et al., 1995).

Among patients with chronic alcoholic pancreatitis, about half have raised glucose concentration and about three quarters have abnormal oral glucose tolerance tests (Andrea Rambaldi et al., 1995). Unlike patients with type 2 diabetes, patients with chronic alcoholic pancreatitis do not have raised insulin release after an oral GTT and the impaired insulin secretion due to a loss of beta cell mass in the pancreas. Further more such patients often demonstrate insulin resistance (Cavallini and Frulloni, 2001).

DM in alcoholic cirrhosis:

Insulin is subjected to a high first pass metabolism in the normal liver, with about 50% being extracted (Nygren et al., 1985). With decreasing liver function and increasing intra hepatic and extra hepatic portal-systemic shunting of cirrhosis, the
fractional hepatic extraction of insulin is significantly decreased to about 13% (Nygren et al., 1985).

Accordingly cirrhotic patients have about six times higher peripheral fasting insulin concentration than normal controls (Kruszynska et al., 1998). This hyperinsulinemia is found irrespective of the development of diabetes. Despite the hyperinsulinemia, the blood glucose concentration is generally higher than normal after glucose loading, implying decreased insulin sensitivity in alcoholic cirrhosis (Yadav et al., 2007). Moreover alcoholic cirrhotics display changes in maximum insulin secretory capacity rather than altered beta cell sensitivity to glucose (Kruszynska et al., 1998). Compared to normal controls, non-diabetic alcoholic cirrhotics have significantly increased maximum insulin secretion, whereas diabetic alcoholic cirrhotics demonstrate impaired maximum insulin secretion.

In conclusion, glucose intolerance in alcoholic cirrhotics results from impaired insulin secretion and insulin resistance.

**Vitamins and Proteins:**

Those who use alcohol excessively deprive their bodies of essential nutrients. The drinker and the recovering alcoholic must pay special attention to diet. A diet high in protein not only provides many of the nutrients vital to recovery, but also keeps the blood sugar from too rapid change. It is better for those who drank excessively to get protein from eggs, milk, or vegetables, than from meats or cheeses. Because of an already-fatty liver, excessive drinkers cannot process the extra fat. When they eat meat, fruit should be eaten; it aids in breaking down fats. Vitamin supplements are helpful for people with drinking problems; these include, vitamins A, B, C, and E. Protein supplementation may be important to reducing alcohol craving and maintaining emotional balance for alcoholics wanting to recover from their past heavy drinking. Similarly, a diet high in complex carbohydrates stabilizes blood glucose and reduces the low blood sugar state that can lead to craving alcohol. Understanding one's own special nutritional needs is an important aspect of recovery from excessive alcohol use (Norton et al., 1987; Estruch et al., 1993; Palliyath and Schwartz, 1993; Victor, 1994; Monforte et al., 1995; Pessione et al., 1995).

In patients who are undernourished and have a history of frequent relapse and self-neglect, 200-300 mg thiamine a day over three months or longer is prescribed to minimize the risk of damage to the brain and peripheral nervous system. Oral vitamins...
are absorbed poorly during the early stages of detoxification, so parenteral thiamine may be needed. If the patient is suspected to have or be developing Wernicke's encephalopathy, urgent treatment in hospital with parenteral thiamine is needed.

Other drugs are rarely necessary. Antacids will help relieve stomach pains. Anticonvulsants are of little value in preventing withdrawal fits, and the management of alcohol dependent people with established epilepsy is best supervised by a specialist clinic. Antidepressants are not indicated at this stage in treatment, and antipsychotics are needed rarely.

OTHER DRUGS AND ALCOHOL

Drugs such as marijuana and cocaine which are used like alcohol, for "recreational" purposes have different, but similarly harmful, physical effects. Driving after using either alcohol or marijuana is unsafe, after using both, driving is more than twice as dangerous. Judgment, reaction time, and coordination are worse than with either drug taken alone. Cocaine, "Crack" and amphetamines are fast-acting stimulants. People who use alcohol and stimulant drugs together will drink more to feel the effects of alcohol because of the stimulant effects. When stimulant effects wear off, the alcohol effects "catch up" quickly, and that can be extremely dangerous, both in terms of physical effects and distortions of perception and judgment.

PRESCRIPTIONS

Drugs prescribed for medical conditions are frequently harmful if combined with alcohol. Addiction to alcohol is addiction to all sedatives. Drugs which are prescribed to combat anxiety include various sedatives, "tranquilizers" and barbiturates, most frequently prescribed is Valium. Tranquilizers are addictive, and, if taken with alcohol will multiply the effects of both to sedate the user. This interactive effect can lead to a coma or death. Sometimes antidepressants, or amphetamines, are prescribed to treat depression or for weight control. These drugs speed up the nervous system and are addicting. Because they are stimulants, the effects of drinking while using them is like the effect of cocaine with alcohol -- they "cancel each other out" until the stimulant wears off, then intoxication occurs quickly.

Medication of any kind should not be mixed with alcohol. None should be taken by the recovering person, unless the physician who prescribes is fully aware of the alcohol use history.
First, alcoholics need to detoxify their body by getting the alcohol out of their system. That typically involves three to four days, most often done in a hospital. Then it is followed by counseling, either in a group support program or private counseling. New drugs are available such as Naltrexone, an opiate blocking medication that comes in pill form or a longer lasting once a month injection called Vivatrol. Alcohol stimulates a pleasure center in the brain and releases beta-endorphins that create that buzz or high. Naltrexone blocks the endorphin receptors in the brain so the person doesn't get the same pleasure from drinking. There are other drug options to help cut craving. Medication plus counseling can be very effective.

**ALCOHOL ABUSE**

Alcohol abuse differs from alcoholism in that it does not include an extremely strong craving for alcohol, loss of control over drinking, or physical dependence. Alcohol abuse is defined as a pattern of drinking those results in one or more of the following situations within a 12-month period:

- Failure to fulfill major work, school, or home responsibilities;
- Drinking in situations that are physically dangerous, such as while driving a car or operating machinery;
- Having recurring alcohol-related legal problems, such as being arrested for driving under the influence of alcohol or for physically hurting someone while drunk; and
- Continued drinking despite having ongoing relationship problems that are caused or worsened by the drinking.

**1.9 REHABILITATION AND COUNSELING FOR ALCOHOLICS**

Drug rehabilitation (often drug rehab or just rehab) is an umbrella term for the processes of medical and/or psychotherapeutic treatment, for dependency on psychoactive substances such as alcohol, prescription drugs, and so-called street drugs such as cocaine, heroin or amphetamines. The general intent is to enable the patient to cease substance abuse, in order to avoid the psychological, legal, financial, social, and physical consequences that can be caused, especially by extreme abuse. Psychological dependency is addressed in many drug rehabilitation programs by attempting to teach the patient new methods of interacting in a drug-free environment. In particular,
patients are generally encouraged or required not to associate with friends who still use the addictive substance.

Various types of programs offer help in drug rehabilitation, including residential treatment (in-patient), out-patient, local support groups, extended care centers.

1.10 VAILANKANNI WARD AND DE-ADDICTION CENTER

Vailankanni ward and de-addiction center: The center was initially under Prajna counseling center before Father Muller's Hospital took over. The programme primarily focuses on providing residential and supportive treatment services for the rehabilitation of alcoholics. Over years the De-addiction center gained a stable platform under the roof of Vailankanni Ward. It has resident facilities for 60 persons Medical, psychiatric and nursing care is being looked after without any extra cost to the hospital At present the center has a full fledged ward only for alcoholic men. The dire need of the center is to open a separate full fledged ward for women alcoholics

Vailankanni ward and De-addiction center follows a combination of models with special emphasis on the community-oriented therapeutic model. The programme follows a multi-disciplinary, holistic and integrated approach. The team consists of psychiatrists, psychologists, family therapists, medical specialists, counselors, social workers, after-care workers, recovered alcoholics, community workers, nurses and yoga therapists

Integrated community-based eclectic approach:

This consists of intake motivational counseling, de-toxification, individual counseling, group counseling / psychotherapy, educational sessions, behavior modification, marital counseling, weekly family meetings, monthly family get-togethers, birthday concept, relapse of prevention programme, Al-Anon (family) and Al-Teen (children of alcoholics) meetings. The “Al-teen” which is a fellowship of young children, whose lives have been affected by an alcoholic member, provides a forum to share their experiences, fears, worries. Battered wives and children are provided with care and protection.

Vailankanni ward as a unique de-addiction cum rehabilitation center in Dakshina Kannada District, has completed 12 years of its service and since its
inception has successfully treated over 12,000 alcoholics. A total of 3528 addicts underwent treatment for the second time.

Generous contributions of Aubrey D'Souza Charitable foundation and Mrs Benny Pinto have initiated the establishment of Vailankanni Ward. It has been successfully reaching out to help men, women and family in great distress. This good work would be carried forward in the coming years to a wide spectrum of society in different dimensions.