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Pharmacological research and review project on the effect of curcumin on weight loss

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Introduction

Turmeric and Weight Loss-

Obesity is an increasing social and medical problem all over the world and refers to having too much body fat. However , it is not like being overweight which refers to weighing too much . A person may be overweight because of extra muscle or bone or water retention in the body. Both result in a person having more than the recommended healthy body weight . This could cause a host of medical problems like osteoarthritis , gallstones,liverproblems ,heart diseases ,high blood pressure ,high triglycerides, high cholesterol ,diabetes sleep , apnea etc. Apart from eating too much,obesity can be caused by excess alcohol consumption ,menopause , hypothyroidism , not exercising etc. The first step to fighting obesity or being overweight is to start a weight loss program .This includes following a strict diet ,exercise and in some cases even surgery . The liver is organ that is essential for fat burning .Studies have found that when the liver is gets damaged ,the detoxification process is reduced. Turmeric can help detoxify the liver and protect the cell damage caused due to environmental ,attack from free radicals etc.High cholesterol causes plaque build upin arterial walls leading to coronary heart diseases ,athersclerosis,weight gain etc.

Research found that turmeric can extracts can lower blood cholesterol level –especially , LDL ‘bad ‘ cholesterol . It has lipid lowering properties. This can reduce cholesterol levels and benefits weight loss by reducing adipose tissue weight gain .Thromobogenesis is the process that takes places when fat is burned by the central nervous system in order to maintain body temperature.

The liver responds to injury by wound healing and subsequently, fibrosis.This response is after essentially all kinds of injury (whether virus,alcohol, or other) and ultimately leads to cirrhosis in some patient. The observations that any of several types of liver diseases and their injury result in cirrohsis suggests common pathogenesis. It is now regonised that a population population of effector cells play a critical role in the fibrogenic process.A classic effector cell,the hepatic stellate cell ,is one of the most important fibrogenic

cells in the liver. This cell undergoes a transformation during injury, termed "activation". The activation process is complex, but one of its most prominent features is the synthesis of large amounts of extracellular matrix, resulting in deposition of scar or fibrous tissue. Thus, the hepatic stellate cell and /or other fibrogenic process is dynamic and that even advanced fibrosis is reversible .The best curcuminoid, a group of phenolic compound isolated from roots of curcuma longa (zingiberaceae),exhibit a variety of effects on health and on events that help in preventing certain diseases. A vast majority of these studies were carried out with curcumin ,which a major curcuminoid. The most detailed studies using curcumin include anti-inflammatory, antioxidant, anti -carcinogenic ,antivirus newer and selective method for the isolation of curcuminoids from turmeric.

In addition, the wound healing and detoxifying properties of curcumin have also received considerable attention. As a result of extensive therapeutic properties of curcumin there is a need to develope.

Objective-

1. To heal many health disorders like liver problems , digestive disorders, treatment for skin diseases and wound healing turmeric has long been used in medicinal as anti-inflammatory .
2. Turmeric is considered as a digestive bitter and a carminative.
3. Turmeric improves the body's ability to digest fat.
4. Turmeric can be useful to treat the liver conditions such as hepatitis, cirrhosis and jaundice.
5. Turmeric makes cholesterol level low and inhibited the oxidation of low density lipoprotein (LDL).

Anatomy of the Liver-

The liver is the largest organ of the body, weighing 1 to 1.5 kg and representing 1.5 to 2.5% of the lean body mass. The size and shape of the liver vary and generally match the general body shape-long and lean or squat and square.

The liver is located in the right upper quadrant of the abdomen under the right lower rib cage against the diaphragm and projects for a variable extent into the left upper quadrant.

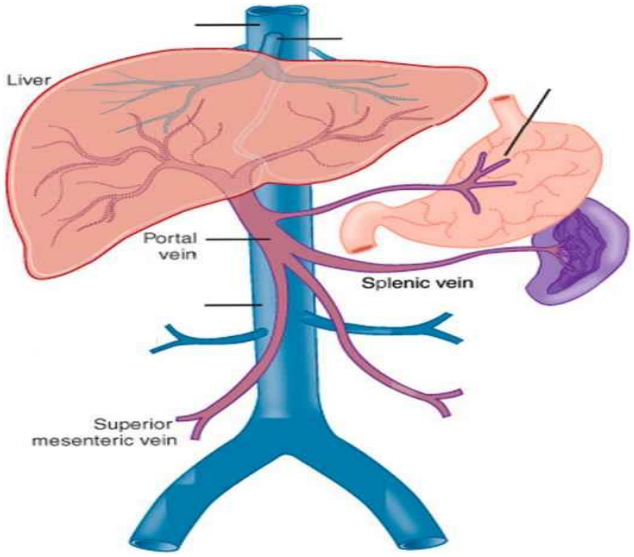

Figure 1 :structure of liver

Histology-

The majority of cells in the liver are hepatocytes, which constitute two-thirds of the mass of the liver. The remaining cell types are Kupffer cells, stellate (Ito or fat-storing) cells, endothelial cells and blood vessels, bile ductular cells, and supporting structures. Viewed by light microscopy, the liver appears to be organized in lobules, with portal areas at the periphery and central veins in the center of each lobule. However, from a functional point of view, the liver is organized into acini, with both hepatic arterial and portal venous blood entering the acinus from the portal areas (zone 1) and then flowing through the sinusoids to the terminal hepatic veins (zone 3) the intervening hepatocytes constituting zone 2. The advantage of viewing the acinus as the physiologic unit of the liver is that it helps to explain the morphologic patterns and zonality of many vascular and biliary diseases not explained by the lobular arrangement. Portal areas of the liver consist of small veins, arteries, bile ducts, and lymphatics organized in a loose stroma of supporting matrix and small amounts of collagen. Blood flowing into the portal areas is distributed through the sinusoids, passing from zone 1 to zone 3 of the acinus and draining into the terminal hepatic veins ("central veins"). Secreted bile flows in the opposite direction, in a counter current pattern from zone 2 to zone 1. The sinusoids are lined by unique endothelial cells that have prominent fenestrae of variable size, allowing the free flow of plasma but not cellular elements. The plasma is thus in direct contact with hepatocytes in the subendothelial space of Disease. Hepatocytes have distinct polarity. The basolateral side of the hepatocyte lines the space of Disse and is richly lined with microvilli, it demonstrates endocytotic and pinocytotic activity, with passive and active uptake of nutrients, proteins, and other molecules. The apical pole of the hepatocyte forms the cannicular membranes through which bile components are secreted. The canniculi of hepatocytes form a fine network, which fuses into the bile ductular elements near the portal areas. Kupffer cells usually lie

within the sinusoidal vascular space and represent the largest group of fixed macrophages in the body. The stellate cells are located in the space of disse but are not usually prominent unless activated, when they produce collagen and matrix. Red blood cells stay in the sinusoidal space as blood flows through the lobules, but white blood cells can migrate through or around endothelial cells into the space of disse and from there to portal areas, where they can return to the circulation through lymphatics. Hepatocytes perform numerous and vital roles in maintaining homeostasis and health. These functions include the synthesis of most essential serum proteins (albumin, carrier proteins, coagulation factors, many hormonal and growth factors), the production of bile and its carriers (bile acids, cholesterol, lecithin, phospholipids), the regulation of nutrients (glucose, glycogen, lipids, cholesterol, amino acids), and metabolism and conjugation of lipophilic compounds (bilirubin, anions, cations, drugs) for excretion in the bile or urine. Measurement of these activities to assess liver function is complicated by the multiplicity and variability of thesefunctions.The most commonly used liver "function" tests are measurements of serum bilirubin, albumin, and prothrombin time. The serum bilirubin level is a measure of hepatic conjugation and excretion, and the serum albumin level and prothrombin time are measures of protein synthesis. Abnormalities of bilirubin, albumin, and prothrombin time are typical of hepatic dysfunction. Frank liver failure is incompatible with life and the functions of the liver are too complex and diverse to be subserved by a mechanical pump; dialysis membrane; or concoction of infused hormones, proteins, and growth factors.

Functions of the Liver-

Liver is a very important "way station" in the blood's journey throughout your body. All of the blood leaving the stomach and intestines passes through the liver. But why exactly does your liver need the oxygen and nutrients from your blood? What does it do? Some people think of the liver as the body's chemical plant and inspection station.

For a chemical to be eliminated from the body at a site of elimination (i.e., kidney) that is distant from the site of storage (i.e., adipose tissue) or toxicity (i.e., brain), the chemical must be transported from the site of origin to the site of elimination. Chemicals are transported to the site of elimination largely via the circulatory system. Sufficiently water-soluble chemicals can freely dissolve into the aqueous component of blood and be transported by both diffusion and blood circulation to sites of elimination. With decreasing water solubility and increasing lipid solubility, chemicals are less likely to freely diffuse into blood and extraction of these chemicals from sites of toxicity or

Storage can be more challenging. These materials generally associate with transport proteins in the blood, which either contain binding sites for chemical attachment or lipophilic cores (lipoproteins) into which lipophilic chemicals can diffuse. The blood contains various transport proteins that are typically suited for the transport of specific endogenous chemicals. These include albumin, sex steroid-binding globulin, and lipoproteins. Often xenobiotics can utilize these proteins, particularly the nonspecific transporters, to facilitate mobilization and transport in the aqueous environment of the blood. At the site of elimination, xenobiotics may diffuse from the transport protein to the membranes of the excretory organ, or the transport

protein may bind to surface receptors on the excretory organ, undergo endocytosis and intracellular processing, where the xenobiotic is released and undergoes processing leading to elimination.

Monoamine Oxidases-

Monoamine Oxidases (MAO) catalyze the oxidation of monoamines such as dopamine, serotonin and adrenalin. They are found bound to the outer membrane of mitochondria in most cell types in the body. Two subtypes of monoamine oxidase have been identified: MAO-A and MAO-B. Both are found in neurons and astroglia, with MAO-A also found in the liver, gastrointestinal tract and placenta and MAO-B in blood platelets. Abnormal regulation of myosin the body has been associated with depression, substance abuse, attention deficit disorder, and irregular sexual maturation.

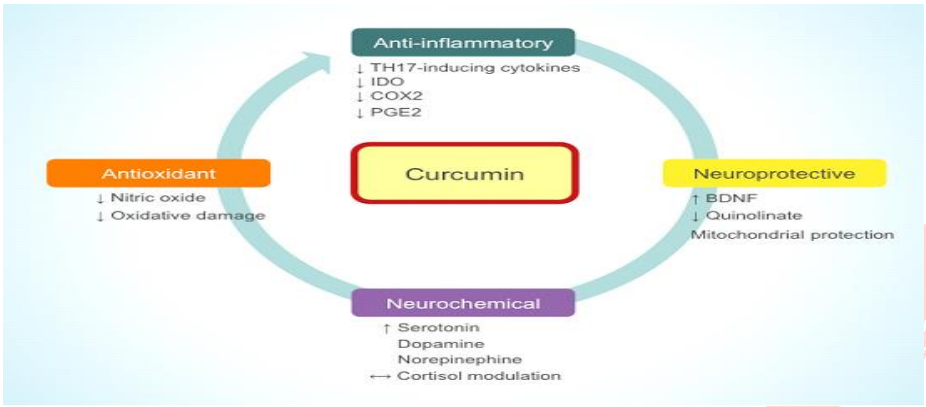

Figure 2: Uses of curcumin

Turmeric Extract (Curcumin) Is An Ideal Weight Loss Supplement-

In the new study titled, "Curcumin induces brown fat-like phenotype in 3T3-L1 and primary white adipocytes," Korean researchers established for the first time that curcumin is capable of inducing browning of white fat cells (adipocytes) through at least four different mechanisms:-

1. By enhancing the expression of brown fat specific genes. This is a form of nutrigenomic "epigenetic modification," which means that a nutrient is capable of altering a cell's patterns of gene expression "from the outside in," as it were, resulting in significant changes in the structure and function of the cells involved.

2. By stimulating the production of new mitochondria, as evidenced by increased activity of the electron transport chain and increased fatty acid oxidation. Mitochondrial biogenesis can be stimulated with other natural substances and therapeutic modalities and is an intervention that may be of special benefit in age-associated loss of muscle and brain function, enhancing athletic performance, and in improving mitochondrial disorders.

3. By increasing protein levels of hormone-sensitive lipase and p-acyl-carboxylase, two markers that play a role in increasing fat-degrading processes (lipolysis) and the suppression of new fat production (lipogenesis).

4. By increasing the activity AMP-activated protein kinase (AMPK). AMPK activity is something of a cellular metabolic master switch that improves metabolic homeostasis, which is often out of balance in overweight and obese individuals.

In addition to these four mechanisms of action contributing to curcumin's brown fat supporting properties, the researchers also noted that because curcumin is a well-established anti-inflammatory agent, and because obesity and its various co-morbid states such as diabetes and cardiovascular disease are conditions that involve upregulated and unremitting inflammatory and/or or dysregulated inflammatory response, curcumin's anti-obesity effects may be in part due to its inflammation-reducing properties.

One additional relevant mechanism of action not discussed in this study, but recently identified in a study published earlier this year in the journal Molecular Medicine Reports, is curcumin's ability to induce programmed cell death (apoptosis) in white fat cells. This may contribute permanently to reducing the overall ability of the body to store urine.

Steatosis (also called fatty change, fatty degeneration, or adipose degeneration) is the process describing the abnormal retention of lipids within a cell. It reflects an impairment of the normal processes of synthesis and elimination of triglyceride fat. Excess lipid accumulates in vesicles that displace the cytoplasm. When the vesicles are large enough to distort the nucleus, the condition is known as macrovesicular steatosis; otherwise, the condition is known as microvesicular steatosis. While not particularly detrimental to the cell in mild cases, large accumulations can disrupt cell constituents, and in severe cases the cell may even burst.

The risk factors associated with steatosis are varied, and include diabetes mellitus, protein malnutrition, hypertension cell toxins, obesity, anoxia and sleep apnea. As the liver is the primary organ of lipid metabolism it is most often associated with steatosis ; however, it may occur in any organ, commonly the kidneys, heart, and muscle.

RENAL ELIMINATION

The kidneys are the sites of elimination of water-soluble chemicals that are removed from the blood by the process of reverse filtration. Two characteristics are primarily responsible for determining whether a chemical will be eliminated by the kidneys: size and water solubility.

Size-The reverse filtration process requires that chemicals to be removed from the blood are able to pass through 70 to 100 A pores. As a rule, chemicals having a molecular mass of less than 65,000 are sufficiently small to be subject to reverse filtration.

Water Solubility-

Non-water-soluble chemicals will be transported to the kidney in association with transport proteins. Thus, in association with these proteins, the chemicals will not be able to pass through the pores during reverse filtration. Lipophilic chemicals are generally subject to renal elimination after the elimination of toxicants have undergone hydroxylation or conjugation reactions in the liver or elsewhere.

Blood is delivered to the human kidney by the renal artery. Blood flows to the

Kidneys of the adult human at a rate of roughly 1 L/min.The adult human kidney contains approximately 1 million functional units, called nephrons, to which the blood is delivered for removal of solutes. Collected materials are excreted from the body in the urine. Blood entering the nephron passes through a network of specialized capillaries called the glomerulus. These capillaries contain the pores through which materials to be eliminated from the blood pass. Blood in the capillaries is maintained under high positive pressure from the heart coupled with the small diameter of the vessels. As a result these sufficiently small solutes and water are forced through the pores of the glomerulus. This filtrate is collected in the glomerular (or Bowman's) capsule in which the glomerulus is located. Included in this filtrate are

Water, ions, small molecules such as glucose, amino acids, urate, and foreign chemicals. Large molecules such as proteins and cells are not filtered and are retained in the blood. Following glomerular filtration, molecules important to the body are re-absorbed from the filtrate and returned to the blood. Much of this re-absorption occurs in the proximal tubules . Cells lining the proximal tubules contain finger like projections that extent into the lumen of the tubule. This provides an expanse of cell surface area across which water and ions can diffuse back into the cells and, ultimately, be returned to the blood. The proximal tubules also contain active transport proteins that recover small molecules such as glucose and amino acids from the filtrate. From the proximal tubules, the filtrate passes through The Loop Of Henle. Significant water vein glomerulus loop of henle capillary bed to the urinary bladder artery glomerular capsule proximal tubules.

The nephron is the functional unit of the kidney that is responsible for the removal of water-soluble wastes and foreign compounds from the blood.

Re-absorption occurs in the descending portion of the loop, resulting in concentration of the filtrate. Water re-absorption does not occur in the ascending portion of the loop. Rather, the remaining, concentrated ions such as sodium, chloride, and potassium are re-absorbed. Those materials retained in the filtrate during passage through the nephron constitute the urine. The urine is transported through the ureters to the bladder and retained until excretion occurs. The kidneys are a common site of chemical toxicity since the nephron functions to concentrate the toxicant and thus increase levels of exposure to the materials. This increased exposure can result from the concentration of the toxicant in the tubules. It also can occur by concentration within the cells of the nephrons when a chemical is capable of utilizing one of the active transport proteins and is shuttled from the lumen of the tubules into the renal cells.

LIVER DISEASES-

Though While there are many causes of liver disease, they generally present clinically in a few distinct patterns, usually classified as hepato-cellular, cholestatic (obstructive),or mixed. In hepato-cellular diseases(such as viral hepatitis or alcoholic liver disease),features of liver injury, inflammation, and necrosis predominate. In (such as gall stone or malignant obstruction, primary biliary cirrhosis, some drug-induced liver diseases), features of inhibition of bile flow predominate. In a mixed pattern, features of both hepato-cellular and cholestatic injury are present (such as in cholestatic forms of viral hepatitis and many drug-induced liver diseases). The pattern of onset and prominence of symptoms can rapidly suggest a diagnosis,

Particularly if major risk factors are considered, such as the age and sex of the patient and a history of exposure or risk behaviours .

Typical presenting symptoms of liver disease include jaundice, fatigue, itching, right upper quadrant pain, abdominal distension, and intestinal bleeding. At present, however, many patients are diagnosed with liver disease who have no symptoms and who have been found to have abnormalities in biochemical liver tests as a part of a routine physical examination or screening for blood donation or for insurance or employment. The wide availability of batteries of liver tests makes it relatively simple to demonstrate the presence of liver injury as well as to rule it out in some one suspected of liver disease.

Evaluation of patients with liver disease should be directed at

(1) establishing the etiologic diagnosis,

(2) estimating the disease severity (grading), and

(3) establishing the disease stage (staging).

Diagnosis should focus on the category of disease, such as hepato-cellular, cholestatic, or mixed injury, as well as on the specific etiologic

Diagnosis. Grading refers to assessing the severity or activity of disease—active or inactive, and mild, moderate, or severe. Staging refers to estimating the place in the course of the natural history of the disease, whether acute or chronic; early or late; pre-cirrhotic, cirrhotic, or end-stage.

The liver injury may take several forms and involve hepatocytes, vascular cells or bile ducts. The most important diseases are:

1. Billiary obstruction
2. Metabolic lesions caused by generic disease or exogenous substance, such as alcohol
3. Inflammation, especially caused by hepatitis virus
4. Cirrhosis
5. Neoplasia

Modern allopathic system of medicine has evolved phenomena only, the remedy for hepatic disease still depends on herbs or herbal medicines. Many herb sand herbal medicines are being used since ages to treat hepatic diseases.

The types of liver disease-

There are many types of liver disease, three of the most common are:

1. Alcohol-related fatty liver-disease-

Where the liver is damaged after alcohol abuse.

2. Non-alcoholic fatty liver disease-

A build-up of fat within liver in liver cells.

3. Viral (Hepatitis)-

An inflammation(swelling) of the liver caused by a viral infection.

4. Autoimmune (chronic hepatitis)-

Severe form of hepatitis where blood cells attack and destroy liver cells.

All types of liver disease above can cause damage to the liver. The advice on this page is specific to alcohol-related liver disease.

Alcohol-related liver disease can be prevented if you understand the impact excessive alcohol drinking can have on your liver and take-steps to control the amount you drink. For more information on how alcohol affects your health, read about the short and long-term effects of alcohol in your body.

Stages of Liver Disorder-

Even with a wide range of conditions diagnosed as liver disease, the stages and damage to the organ are consistent. From the beginning of the condition to advanced liver disease, the damage progresses in these four stages:

Stage 1: Initial Stage of Liver Disease-

With any condition causing liver disease, the first step includes inflammation of the liver or bile duct. This inflammation causes abdominal pain as the body tries to fight the infection or irritation. If left untreated, this inflammation can cause damage to the liver, making the condition worse. In this beginning stage, unlike some conditions in advanced liver disease, the symptoms and inflammation is treatable to prevent the second step of the disease.

Stage 2: Fibrosis of the Liver-

Many times, symptoms of liver disease aren't present until this stage or the next. In the fibrosis stage, damage or scarring from the first stage begins to block the normal blood flow of the liver. In this stage, the liver isn't functioning correctly, but through treatment, it may be able to heal and prevent any further progression of the disease.

Stage 3: Cirrhosis of the Liver-

A chronic condition, cirrhosis of the liver creates permanent scarring that blocks the blood flow. This dangerous stage causes other serious conditions and symptoms that increase the severity of the liver disease and is recognized as one of the leading causes of death in the US. For this stage of the disease, doctors focus treatment on managing the symptoms in order to prevent the most advanced liver disease stage.

Stage 4: Liver Failure and Advanced Liver Disease-

In the final stage of the disease, liver failure signals the end of all normal liver function. The patient now requires immediate medical attention to prevent death. Symptoms of liver failure include vomiting, diarrhea and fatigue as well as the symptoms from stage 3. While the progression from cirrhosis to failure can take years, the damage is irreversible and leads to eventual death.

The key to treating liver disease is to diagnose the condition as early as possible. If you experience any of the symptoms in these stages, seek medical help immediately.

Steatosis (also called fatty change, fatty degeneration, or adipose degeneration) is the process describing the abnormal retention of lipids within a cell. It reflects an impairment of the normal processes of synthesis and elimination of triglyceride fat. Excess lipid accumulates in vesicles that displace the cytoplasm. When the vesicles are large enough to distort the nucleus, the condition is known as macro-vesicular steatosis ; otherwise, the condition is known as micro-vesicular steatosis. While not particularly detrimental to the cell in mild cases, large accumulations can disrupt cell constituents, and in severe cases the cell may even burst.

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Cholestaticdiseases-

Primary biliary cirrhosis is the gradual destruction of the biliary system for unknown .Although the cause of this serious condition is not known, it has many features to suggest tshat it is an autoimmunedisease. Autoi mmunity describes the process whereby the body's defense mechanisms are turned against itself. Theimmun e system is supposed to recognize and attack only dangerous foreign invaders like germs, but many times ita ttacks, for no apparent reason, the cells of the body itself. Autoimmune reactions occur in many different tis sues of thebody, creating a great variety of diseases.

Primary biliary cirrhosis progressively destroys the system that drains bile from the liver into the intestines. Bile is acollection of waste products excreted by the liver. As the disease progresses it also scars the liver, le ading to cirrhosis.In some patients, the disease destroys the liver in as little as five years. In others, it may lie dormant for a decade ormor

Causes and symptoms-

Ninety percent of patients with this disease are women between the ages of 35 and 60. The first sign of it ma y be anabnormal blood test on routine examination. Itching is a common early symptom, caused by a buildu p of bile in the skin.Fatigue is also common in the early stages of the disease. Later symptoms include jaund ice from the accumulation ofbile and specific nutritional deficiencies.

Bruising from vitamin K deficiency, bone pain from vitamin D deficiency, nightblindness from vitamin A d eficiency, and skin rashes, possibly from vitamin E or essential fatty acid deficiency.

All these vitamin problems are related to the absence of bile to assist in the absorption nutrients from the intestines.

Billiary obstruction:

Bile flow obstruction results in jaundice. Lesion in the main extra hepatic bile duct, such as carcinoma, impacted bile stone or sclerosing cholangitis typically cause obstructive jaundice. Prolong bile duct obstruction may cause secondary biliary cirrhosis.

Metabolic disorder:

Metabolic disorder of the liver may be hereditary (genetic) or acquired.

Representative hereditary hyperbolic rubinemias and disturbances involving the intermediate metabolism of lipids, carbohydrates, proteins and heavy metal are few example of metabolic disorder of liver.

Congenital metabolic disorder:

Congenital hyper bilirubinemia occurs in several forms. The best known congenital jaundice syndromes are Gilbert, Rotor syndrome and Dubin-Jonshon syndrome. Genetic enzyme deficiencies such as alpha-1-antitrypsin deficiency may also result in liver injury, which ultimately leads to cirrhosis.

Acquired metabolic disorder:

Metabolic disorder can be induced in liver cells by a variety of ingested substances such as toxins, drugs, foods and beverages. Alcohol produces three types of liver disease such as hepatomegaly, alcoholic hepatitis and cirrhosis. Several drug for example methyldopa, nitrofurantoin , isoniazide , ketokonazole and acetaminophen , etc., can induce hepatitis.

Viral hepatitis:

Acute viral hepatitis is a systemic infection manifested primarily by an acute attack on the hepatocytes. Five hepato-tropic viruses have been identified (HAV, HBV, HCV,HDV, HEV). Hepatitis A (HAV) causes acute, self-limited disease that is transmitted orally. Hepatitis B (HBV) and Hepatitis C virus (HCV) are transmitted by exchange of body fluids such as through blood transfusion or sexual contacts. Hepatitis D(HDV) is a viroid that causes inflammation only in concrete with HBV. Hepatitis E virus is transmitted by enteric route and cause self limited disease. Chronic hepatitis is an uncommon but important complication of HBV and combined HBV – HDV infection. The liver injury results from an inflammatory immune attack against hepatocytes.

Cirrhosis:

Cirrhosis is a chronic liver disease characterized by wide spread fibrosis and

Regenerative nodule, which diffusely replace the normal liver parenchyma. The major causes of cirrhosis are alcoholism and viral hepatitis (HBV, HCV and HDV).

Liver tumor:

Primarily liver tumour may originate from liver cells, from bile ductules and less often from kupffer cells and connective tissue cell of hepatic capsule and portal tracts. Hepatocellular carcinoma (malignant

hepatonia) is the most common primary malignant liver tumor. Cholangio cellular carcinoma is a malignant tumor of bile ducts.

Necrosis:

Hepatic necrosis may be zonal massive or diffuse. Zonal necrosis may be in the central, peripheral or midzone of the lobule, depending on the agent. In general, the necrosis that is produced by intrinsic (predictable) hepato-toxins is zonal; while that produced by an idiosyncratic reaction to a drug is usually diffuse or massive. Centrizonal necrosis is the characteristic lesion produced by carbon tetrachloride, chloroform, iodo form, bromobenzene and other series of acetaminophen.

Degeneration:

Agents that lead to necrosis also lead to degeneration of hepatocytes that is evident from the fact that, prior to the development of necrosis or at its periphery, hepatocytes show sub-necrotic damage that includes hydrophilic degeneration or balloning and eosinophilic degeneration , the latter leading to the free sinusoidal, acidophilic bodies.

Fatty Liver:

Fatty liver refers to the abnormal accumulation of fat in hepatocytes. At the same time there is a decrease in plasma lipids and lipoproteins. Many toxicants may cause lipid accumulation in the liver, but their mechanisms differs. Basically lipid accumulation is related to disturbances in either the synthesis or the secretion of lipoproteins. Excess lipid can result from an oversupply of free fatty acids from adipose tissues or more commonly, due impaired release of triglycerides from the liver into the plasma. Triglycerides are secreted from the liver as lipoproteins (very low density lipoprotein, VLDL). There are number of points at which this process can be disrupted. Some of the more important ones are interference with synthesis of the protein moiety, impaired conjugation of triglyceride with lipoprotein and interference with transfer of VLDL across cell membranes.

Triglyceride cycle in the pathogenesis of fatty liver“=” are metabolic blocks.

Steatosis:

It is a form of fatty liver disease caused by agents like tetracycline, alcohol, Methotrexate etc. Two main types of fatty changes that occur are micro-vascular steatosis and macro vascular steatosis. In micro-vascular steatosis (caused by tetracycline and number of experimental toxins), the hepatocytes are filled with tiny droplets of fat that do not displace the nucleus. In macro-vascular steatosis (caused by alcohol, methotrexate etc), most of fatty cells contain a large droplet of fat which displaces the nucleus to the periphery.

Curcumin in turmeric can fasten to capsicain receptors and increases thrombogenesis rates .This can leads to greater fat burning and helps with weight loss .

In vivo and in vitro or weight loss through angiogenesis new blood vessel growth and reducing fat contents in fat cells have been conducted.

Adipose tissue which store fat cells store fat cells require angiogenesis required nutrients and oxygen to adipocytes (fat cells) .

PHARMACOGNOSTIC ACCOUNT

TURMERIC-

Figure 3: Turmeric Plant

Synonyms-

curcuma, turmeric, rhizome curcumae

Biological source-

Turmeric consists of dried prepared rhizomes of Curcuma longa.

Family-

Zingiberaceae

Geographical source-

Turmeric is indiogenous to South Asia .It is cultivated on a larger scale in India, Pakistan, China , Malaya and other tropical countries .

Chemical constituents –

- Turmeric rhizomes contain about 5 % curcuminoids consisting of a mixture of compounds known as curcumin and its derivatives .
- These compounds are diaryl heptanoid compounds of dark yellow colour.
- The standardized extracts of curcumin generally consists of curcumin, de-methoxy curcumin .

- It also contains dicaffeoyl methane and caffeoyl feruloyl methane .
- The essential oil is present to the extent of 5 % and constituents about 25 % zingiberine as a major constituents along with sesquiterpene ketone , turmerone sesquiterpenes and monoterpoinds .
- Mono saccharides such as glucose (28 %) , fructose (12%) and arabinose are other compounds present in the rhizome.
- It consists of abundant starch grains .

Microscopical characters-

The transervse section of turmeric rhizomes shows shows thicker pale brown cork and tubular polygonal epidermal layer ,followed by cortical region , and then endodermis and stele at the centre.

The vascular bundles of the prepared turmeric rhizomes are mostly full of gelatinized starch.

Uses-

- Turmeric is used since centuries as a condiment and colouring agent.
- The drug has long been used in various traditional systems of medicine like Ayurveda , Unani and Chinese medicine .
- It is used as a billiary stimulant , stomachic and to relive menstrual pain.
- Curcumin shows strong antimicrobial ,anti-inflammatory and anti-arthritic activities .
- Recent research findings indicate the potential of turmeric in herbal cosmetics ,as a wound –healing agent and as a potential integrase enzyme inhibitor of the HIV virus .
- Among the other non pharmaceutical uses ,turmeric paper is used the test for boric acid ,which develops a green colour.

Figure 4 : Turmeric Powder

Methodology-

1. Collection and extraction of whole plant of turmeric -

A) Collection:

The whole plant of turmeric was collected from the hilly areas of

Nagava , Sangli , Maharashtra .

B) Authentication:

The collected material was authenticated by Dr. Aditya Arvindekar Sir, Department Of Pharmacognosy.

C) Organoleptic evaluation of the whole plant:

Colour : dark yellow

Odour : Acrid

Taste: Bitter

2. Extraction:

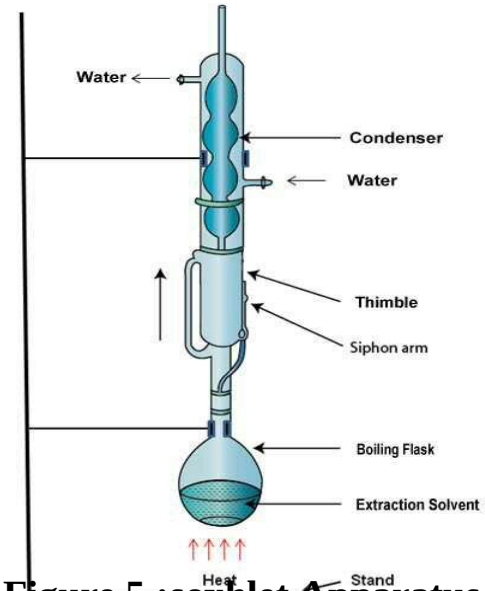

Figure 5 :soxhlet Apparatus

I) Alcoholic (ethanolic) extract :

One Kilogram of whole plant of Curcuma longa were taken and shade

Dried and were coarsely powdered. The powdered materials were exhaustively.

Extracted with 90% ethanol in a soxhlet apparatus. The extracts were concentrated. Using rotary flash evaporator. The obtained residues were dried in a desiccators. The average yield of ethanolic extract was found to be 45 gm

Respectively.

Ii) Aqueous extract:

Shade dried whole plant were mechanically powdered and extracted with distilled water by hot maceration process at 40-60 0C for about 36 hours. Then the aqueous extract was concentrated by vacuum evaporated to powdered form. The extract was re-dissolved in double distilled water just before administration. The average yield of aqueous extract was found to be 20 gm respectively. Percentage of extracts and physical characteristic of extracts of Curcuma longa .Solvent Colour and consistency Percentage yield (w/w).Ethanol Dark brownish and sticky 4.5 %,Aqueous Pale Brownish 2%

2) Tests for Proteins:

A. Biuret test (General test):

3 ml test solution was added to 4% NaOH and few drops of 1% cuso4 solution added. Violet colour was observed.

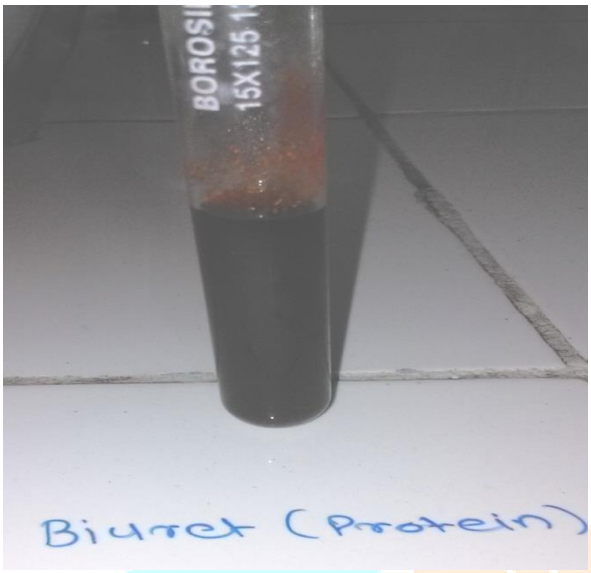

Figure 6: Biuret Test

A2 Test for protein containing sulphur:

5 ml test solution was mixed with 2 ml 40% NaOH and 2 drops 10% lead acetate solution was added. Solution was boiled and it turns into brownish colour.

Tests for Glycosides:

B1 Tests for Cardiac Glycosides:

Baljet's test:

The test solution turn into orange colour with sodium picrate.

Figure 7 : Baljet Test

Legal's test (For cardenoloids):

To 1-2 ml of test solution, added 1 ml pyridine and 1 ml sodium nitroprusside and red colour was observed.

Figure 8: Legal Test

Tests for Alkaloids:

Dragendorff's test:

To 2-3 ml test solution few drops Dragendorff's reagent was added and orange brown precipitate was observed.

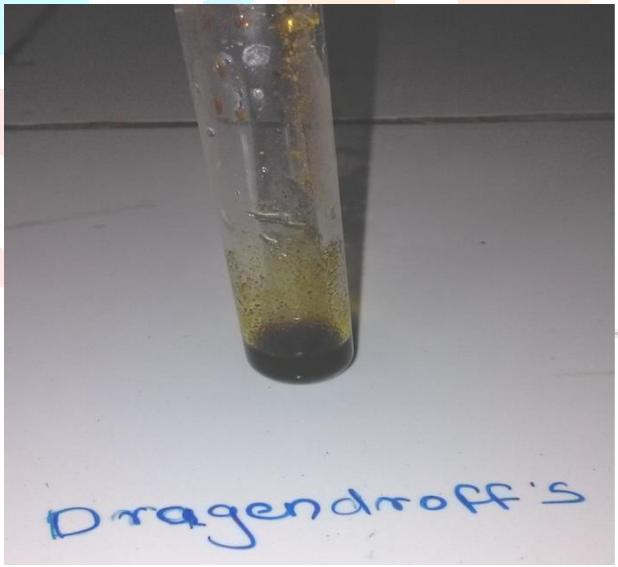

Figure 9: Dragendroffs Test

Hager's test:

To 2-3 ml test solution, few drops of Hager's reagent were added

And yellow precipitate was observed.

Figure 10 : Hagers Test

Wagner's test:

To 2-3 ml test solution, few drops of Wagner's reagent was added and reddish brown precipitate was observed.

Figure 11 : Wagner's Test

The biochemical Parameters estimated includes:

- Serum glutamate oxalo acetate transaminase (SGOT)
- Serum glutamate pyruvate transaminase (SGPT)
- Serum alkaline phosphatase (ALP)

Reagent composition (when reconstituted as directed):

Reagent-1: SGOT Reagent

2-oxaloglutarate	12 mmol/ L
L-Aspartate	200 mmol/L
MDH	>545 U/L
LDH	>909U/L
NADH (yeast)	>0.18 mmol/L
Tris buffer (ph= 7.8+_0.1at 25C)	80 mmol/ L
EDTA	5.0 mmol/L

Table No. 1 Reagent Composition

Also contains fillers and stabilizers.

Reagent reconstitution: The amount of Aqua-4 Supplied in the kit was added to thereagent-1indicated on the label.

Estimation of serum SGPT /ALT (IFCC method):

ALT is also called Alanine Transaminase (ALT), which is located in cytosol of liver cell. During liver cell inflammation and break down of liver cells, they are released into circulation due to increased permeability of cell membrane. Hence determination of ALT is an index of the extent of liver damage.

ALT catalyses the transfer of amino group from L- alanine to 2-oxoglutarate with the formation of pyruvate and L-glutamate. The pyruvate so formed is allowed to react with NADH to produce L-lactate. The rate of this reaction is monitored by an indicator reaction coupled with LDH in the presence of NADH (nicotinamide adenine dinucleotide). The oxidation of NADH in this reaction is measured as a decrease in the absorbance of NADH at 340 nm, which is proportional to SGPT activity.

L-Alanine + 2-Oxoglutarate Pyruvate + L-Glutamate

Pyruvate + NADH L-Lactate + NAD

ALT: Alanine amino transferase

LDH: Lactate dehydrogenase

ALT

LDH

Reagent composition (when reconstituted as directed):

Reagent1: SGPT Reagent

L-Alanine 500mmol/L

NADH (YEAST) 0.18mmol/L

LDH ≥1820IU/L

2- Oxoglutarate 12 m mol/L

Tris- buffer (ph7.5± 0.1 at 250 c) 80 m mol/L

Also contains non-reactive filters and stabilizers.

Reagent reconstitution: The amount of Aqua-4 Supplied in the kit was added to thereagent-1indicated on the label.

Assay Procedure:

Allow the working reagent to attain 370c performing the test. Mix well and aspirate.

Clinical Significance:

ALT is present in high concentration in liver and to a lesser extent in kidney, heart, skeletal muscle, pancreas, spleen and lungs.

Increases:

Increased levels are generally a result of primary liver diseases such ascirrhosis's carcinoma, viral or toxic hepatitis & obstructive jaundice.

Pipette Volume-

Working reagent -1000µl

Test-100 µl

Decreases:

Decreased levels may be observed in renal dialysis patients and those with vit.B6 deficiency

Estimation of serum alkaline phosphate (ALP)64:

Serum alkaline phosphatase hydrolyses p-nitrophenyl phosphate into p-nitrophenol and phosphate in the presence of oxidizing agent Mg2+. This reaction is measured as absorbance is proportional to the ALP activity.

P-Nitro phenyl phosphate + H2O P-Nitrophenol + Phosphate

Reagent composition:

(when reconstituted as directed)

Reagent1: ALP Reagent

P- Nitrophenyl Phosphate 16 m mol/L

Mg2+ 4 m mol/L

Tris /Carbonate buffer (ph10.2 ± 0.2 at 250c) -

Also contains non-reactive filters and stabilizers.

Reagent reconstitution:

The amount of Aqua-4 Supplied in the kit was added to thereagent-1indicated on the label.

Assay Procedure:

Allow the working reagent to attain 370 c performing the test.

Pipette Volume

Working reagent -1000µl

Test -20 µl

Mix well and aspirate.

ALP

Clinical Significance:

ALP is present in high concentration in liver, bone, placenta, intestine and certain tumors.

Increases:

Increased levels of the enzyme occur in liver diseases, bone diseases(Rickets, Paget's disease), hodgkins disease or congestive heart failure.

Decreases:

Decreased levels occur in hypophosphatasia and malnourished patients.

RESULT

The curcumin content of turmeric rhizomes collected from sangali region where determined using UV-Spectroscopy.

The result of preliminary phytochemical evaluation of turmeric rhizomes collected .The result indicated presence of carbohydrate, proteins and alkaloids.

The curcumin group also had lower triglyceride, fatty acids, blood glucose, liver fat and blood cholesterol levels.

This demonstrated that turmeric may benefit weight loss programs.

Curcumin in turmeric can fasten to capsaicin receptors and increase throbmogenesis rates. This leads to greater fat burning and helps with weight loss.

CONCLUSION

From the human volunteer study it is concluded that after taking the curcumin the cholesterol and fat level get decreased which is helpful for weight loss.

From the above study study it was found that turmeric extract can lower blood cholesterol level – especially LDL 'bad' cholesterol. It has lipid lowering properties . This can reduce cholesterol level and benefit weight loss by reducing adipose tissue weight gain.

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